U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-289 817

Health Effects Associated with Diesel Exhaust Emissions, Literature Review and Evaluation

Syracuse Research Corp, NY

Prepared for

Health Effects Research Lab, Research Triangle Park, NC

Nov 78

TECHNICAL REPORT DATA

(Please read Instructions on the reverse before completing)

1. REPORT NO.
EPA-600/1-78-063

2.

3. RECIPIENT'S ACCESSION NO.
PB289817

4. TITLE AND SUBTITLE
HEALTH EFFECTS ASSOCIATED WITH DIESEL EXHAUST EMISSION
Literature Review and Evaluation

5. REPORT DATE
November 1978

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)
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8. PERFORMING ORGANIZATION REPORT NO.

9. PERFORMING ORGANIZATION NAME AND ADDRESS
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Merrill Lane
Syracuse, New York 13210

10. PROGRAM ELEMENT NO.
1AA601

11. CONTRACT/GRANT NO.

12. SPONSORING AGENCY NAME AND ADDRESS
Health Effects Research Laboratory RTP,NC
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, N.C. 27711

13. TYPE OF REPORT AND PERIOD COVERED

14. SPONSORING AGENCY CODE
EPA 600/11

15. SUPPLEMENTARY NOTES

16. ABSTRACT

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In order to accomplish this objective, the following information on diesel emissions has been reviewed in this document: physical and chemical characteristics; biological effects in animals and man; epidemiologic studies; knowledge gaps; and research needs.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
diesel fuels exhaust gases health toxicology reviews		06 F, T

18. DISTRIBUTION STATEMENT
RELEASE TO PUBLIC

19. SECURITY CLASS *(This Report)*
UNCLASSIFIED

20. SECURITY CLASS *(This page)*
UNCLASSIFIED

21. NO. OF PAGES
165

22. PRICE
PC A08 / MF A01

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

United States
Environmental Protection
Agency

Health Effects Research
Laboratory
Research Triangle Park NC 27711

EPA-600/1-78-063
November 1978

Research and Development

PB 289817

EPA

Health Effects Associated with Diesel Exhaust Emissions

Literature Review and Evaluation

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NATIONAL TECHNICAL
INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

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EPA-600/1-78-063
November 1978

HEALTH EFFECTS ASSOCIATED WITH DIESEL EXHAUST EMISSIONS

Literature Review and Evaluation

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the development and revision of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

Assessment of the current state of knowledge regarding the health effects from diesel exhaust emissions requires a comprehensive review of the available literature on this subject. Such an assessment provides the basis for the identification of research needs necessary to establish an adequate data base for decisions by the Administrator regarding the requirement for emission standards or controls.

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Preface

Engineering tests have shown a significant improvement in fuel economy (25% or greater) in light duty vehicles equipped with diesel engines versus those equipped with gasoline engines. Automobile manufacturers are considering a major program for conversion to diesel engines in the automobile fleet by 1985. Available studies show rather large differences in emissions from diesel engine exhausts as opposed to gasoline engine exhaust. Conversion of a major portion of the automobile fleet to diesel engines may significantly change the ambient concentrations of both regulated and unregulated pollutants, and hence the potential human exposure pattern. Such changes may impact upon public health, and consequently require changes in air quality standards, and/or new emissions standards. An assessment of the current state of knowledge regarding the health effects from diesel exhaust emissions, and the identification of major research needs, are important factors which must be considered by the EPA under the 1977 Amendment to the Clean Air Act.

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1.0 Summary and Conclusions

1.1 Biological Effects

Human exposure to vehicular combustion products has been a matter of public health concern for many years. Increasing utilization of internal combustion engines in the development of industrial civilization has forced our society to live with an increased burden of air pollutants. The prospect that passenger cars equipped with diesel engines will soon represent a significant proportion of new car production raises an important question concerning possible impacts on public health. Recognition of this situation allows the opportunity for evaluation of a major environmental change caused by the introduction of greater quantities and/or types of diesel-derived pollutants. It must be recognized, however, that the diesel engine is by no means the only (or even major) source of the pollutants of primary concern.

The components of automotive emissions which are generally regarded to have greatest toxic potential include carbon monoxide, oxides of nitrogen, aldehydes, hydrocarbons, sulfur dioxide, and particulates. In comparison to the gasoline engine operating with or without a catalytic converter, the diesel produces far greater quantities of carbonaceous particulate material. These particles are of respirable size and have high surface areas, enabling them to adsorb gaseous exhaust products. Among these products are small amounts of irritant gases and, perhaps most significant, a large proportion of the polycyclic organic matter (e.g., benzo[a]pyrene) produced during combustion, some of which are carcinogens. This poses the potential risk of delivery of adsorbed gases into the lung by carrier particles. In turn this may lead to extensive localization of harmful materials in the lung, with the accompanying threat of emphysema and cancer development. In addition, fibrotic changes may occur leading to reduced lung compliance and/or obstruction.

Experimental studies designed to establish whether diesel emissions represent a significant threat to human health have been conducted only infrequently during the past 25 years. Moreover, these studies have not provided definitive and comprehensive insight to the health effects of diesel emissions. While it was recognized that the gaseous emissions from diesel engines are comparable to or lower than for the noncatalyst-equipped gasoline engine, the toxicologic role of increased particulate production remains unclear. Just recently, however, it was reported that organic extracts of diesel particulate contained materials that were mutagenic to histidine-requiring strains of Salmonella typhimurium in the Ames assay. This positive result raises a question concerning the potential carcinogenicity of this material, although it is not known how the effects of widely dispersed diesel exhaust in air may be related to the mutagenic effect of diesel extracts. These mutagens in diesel exhaust included, but were not limited to, the well-known polycylic aromatic carcinogens. Other positive compounds found included direct-acting (i.e., not requiring metabolic activation) frameshift mutagens; probably composed of polar compounds such as substituted polynuclear aromatics, phenols, ethers, and ketones. The strong formal relationship between mutagenesis and carcinogenesis thus may implicate extracts of diesel particulate as a carcinogenic material.

It is also known, however, that extracts of airborne particulate pollutants in urban atmospheres and gasoline engine exhaust are also mutagenic in the Ames assay. Moreover, direct-acting mutagens are found in gasoline engine exhaust (noncatalyst-treated) just as they are found in diesel emissions. In addition, a high incidence of skin cancers has been produced in mice by dermal administration with organic extracts of the particulate exhaust fraction from diesel and gasoline engines, and from extracts of ambient particulate pollutants. However, quantitative differences in potentcy are likely to exist among extracts from these various sources.

Investigators have for many years attempted to show that exposure to automotive emissions may lead to the development of cancer. Indeed, it was shown several decades ago that both diesel and gasoline engine exhaust contain carcinogenic polycyclic aromatic hydrocarbons such as benzo[a]pyrene. Since that time, chronic studies have been initiated which involve the inhalation of total diesel emissions by rats and hamsters. Under the conditions of exposure employed, these experiments have thus far failed to produce tumors of the respiratory tract. On the other hand, evidence of serious damage to the respiratory tissues has been obtained. Rats exposed to diesel exhaust for 20 months displayed extensive particulate accumulations in the lungs, accompanied by vesicular emphysema and beginning interstitial fibrosis. Similar observations were made in hamsters, in addition to the presence of cuboidal metaplasia. It is not known whether this tissue damage in rats and hamsters is reversible or if it may lead to significant shortening of life.

A series of in vivo studies with several animal species inhaling diluted irradiated and non-irradiated diesel exhaust are being conducted by the U.S. Environmental Protection Agency (EPA). The initial subacute exposure studies using relatively high concentrations of diesel exhaust (1:12 dilution) were designed to provide preliminary data on toxic effects and target organs. Animals inhaling diesel exhaust for up to two months were found to have black granular particles in alveolar macrophages, and black pigment in the bronchial and carinal lymph nodes. These observations indicated the existence of clearance mechanisms for diesel particulate.

Among guinea pigs inhaling diesel exhaust several exposure-related changes were seen including, increased pulmonary flow resistance, increased lung weight to body weight ratios, and sinus bradycardia. Microscopic examination of the lungs revealed goblet cell hypertrophy and focal hyperplasia of alveolar lining cells, possibly an early indication of damage to the alveolar wall by diesel exhaust. Neither the reversibility of these lesions, nor the degree of functional impairment which accompanies them has yet been determined. Other changes which EPA investigators observed were biochemical alterations in the lungs of rats, behavioral changes in rats, and increased susceptibility to death by respiratory infection in mice.

The presently available data base does not allow for an accurate comparison to be made between the effects of environmentally realistic concentrations of diesel emissions and catalyst or noncatalyst treated gasoline engine exhaust. Nevertheless, diluted gasoline exhaust produced emphysematous lesions in the lungs of dogs as well as a high incidence of bilateral renal sclerosis in rats. Evidence which is available thus far indicates that the use of an oxidation catalyst with gasoline engines will dramatically reduce the toxicity of resulting emissions. This can be attributed to the substantial reductions realized in the emission of most harmful gaseous components in the catalyst-treated exhaust.

An overall assessment of the public health risks associated with diesel exhaust exposure cannot be based solely on the results of available animal studies. This is partly due to the fact that many areas of concern with respect to the toxicity of diesel emissions remain to be explored. In addition, parallel studies with diesel and gasoline engines have not been conducted which would allow direct comparison of results. The alteration of the environment by increased utilization of diesel engines will probably be due to the relative abundance of particulate matter which is emitted. Protection of the public health will thus be best achieved by examining the potential adverse impact of this component of diesel exhaust.

The ultimate goal in demonstrating that increased utilization of dieselized vehicles is an environmentally acceptable substitute for gasoline engines is to provide reliable epidemiologic evidence which supports this claim. Unfortunately, the previous epidemiologic research which is often used to support the safety of the diesel provides only a limited data base, which is clearly inadequate for developing sound conclusions. It is this fact more than anything else which prevents the formulation of a valid health risk assessment for diesel emissions. Among the more recent occupational mortality and morbidity studies which have been reported, it has not been possible to isolate diesel emissions as a singularly important factor in contributing to the excess deaths and adverse health effects occasionally observed. Several studies involving populations of workers exposed to high levels of diesel emissions are currently being conducted by NIOSH. Results of these investigations should be forthcoming within the next several years. Since large populations that have been exposed to ambient levels of light duty diesel emissions do not yet exist, the possibility of conducting community studies at this time is remote, especially when the intervals necessary for the development of neoplasms in humans after exposure are considered. Thus because of the lack of a broad-based community study or well-controlled investigation of a worker population where quantitative exposure data are available, no definitive judgement regarding diesel emissions can now be made.

There is presently no way to place into perspective the hazards of diesel emissions relative to those of automotive emissions as a whole. In the absolute sense, diesel exhaust is a noxious mixture with the potential to produce serious lung disease, behavioral alterations, biochemical changes, and decrements in pulmonary function. Its risk as a human carcinogen, however, is unquantified. When looked upon in light of what we know regarding the potential health effects of noncatalyst-treated gasoline exhaust, the impact of diesel emissions remains unclear. Presently, there are no data which suggest an increased carcinogenic threat from the substitution of diesel- for gasoline-powered light duty vehicles. Further epidemiologic research must be pursued and studies in laboratory animals conducted to characterize cause-and-effect relationships and exposure-response parameters.

1.2 Physical and Chemical Characteristics

A comparison of well-maintained diesel cars (without emission control) and gasoline cars (with emission control) for regulated vaporous emissions shows that diesel cars emit more hydrocarbons than gasoline cars. With automobiles of comparable size, diesel cars emit twice as much hydrocarbons as gasoline cars in the FTP mode. The difference between the two classes of cars becomes even greater in the SET and FET mode, with diesel cars likely to emit three times more hydrocarbons than gasoline cars. The 1977-79 Federal Standard of 1.5 g/mile for hydrocarbon emissions, however, can be met by diesel cars. With respect to CO, both gasoline and diesel cars have about the same emission rate. In the FTP mode, gasoline cars have higher CO emission rates than diesel cars, and the reverse is true in the FET and SET modes. The 1977-79 Federal Standard of 15 g/mile is met by diesel cars. The NO_x emission for gasoline cars is higher than for diesel cars in all modes. Most diesel and gasoline cars will meet the 1977-80 Standard for NO_x of 2.0 g/mile. If a 0.5 g/mile particulate emission standard is introduced for diesel cars in the future, most cars will be able to meet this standard.

The fuel economy consideration is in favor of diesel cars. Based on combined city/highway estimates, the fuel economy for diesel cars is 33 to 60% better than in corresponding gasoline-powered cars.

In terms of unregulated emissions, diesel cars are higher sources of carbonyls and substantially higher sources of particulate emissions. Although there is uncertainty about benzo[a]pyrene emission rates, it appears from the work of Springer and Baines (1977) that diesel cars emit at least an order of magnitude more benzo[a]pyrene than gasoline cars. The reliability of the BaP data in this work, particularly for gasoline exhaust, can be doubted because of the unreliable sample collection technique. It has been demonstrated by Gross (1972) that an increase of 0.5% CO could cause a 45% increase of PNA emissions. Based on this result one would not expect higher PNA emissions from diesel cars. This aspect of research correlating PNA and CO emission rates should be reinvestigated with newer cars equipped with catalytic converters.

Sulfur dioxide emission rates for diesel cars are substantially higher than for gasoline-powered cars. This is, however, expected because the national average diesel fuel contains 0.23 weight% sulfur compared to 0.03 weight% sulfur for the national average gasoline fuels. Reduction of fuel sulfur will reduce the SO_2 emission rate from diesel cars. The sulfate emission rate for gasoline-powered cars with no air pump in the catalytic system is less than for diesel cars. However, with air pumps, the sulfate emission rates become comparable.

Diesel engines produce more visible smoke than gasoline-powered cars. However, diesel cars are capable of operation within the EPA smoke visibility limit, for the most part, with only brief excursions during rapid throttle movement. Diesel exhaust have more odor than gasoline exhausts and the odor intensity of diesel exhaust may noticeably change during the transient cycle. Interior noise levels are slightly higher with the diesel during acceleration than in gasoline automobiles. The idle noise levels are also higher with diesels compared to gasoline cars.

There is a substantial conflict in the available data base among various authors due primarily to nonuniformity of experimental conditions and uncertainty in the variable experimental parameters. In addition to these conflicts in the data base, nonuniformity of data reporting sometimes makes it difficult to compare results among various investigators.

A number of new suspected carcinogenic compounds, namely, methylene-PNA's and nitro-PNA's, have been reported in gasoline exhausts. Their presence can be expected in diesel exhaust and needs confirmation.

2.0 Introduction

According to an EPA projection it has been estimated that automobiles equipped with diesel engines are likely to increase at a rate of 5% per year and capture up to 25% of the U.S. new car market by 1985. The major factor behind the projected increase in diesel-powered automobiles is the considerable fuel economy for this type of car. Of the total estimated auto fuel consumption during the period 1976-2000, it is projected that gasoline will decrease by about 2% and diesel will increase by about 37% (EHA, 1978). At the present time, diesel-powered motor vehicles (mostly heavy duty) contribute about 1% of all motor vehicle emissions (National Academy of Sciences, 1976). With the steady increase of diesel-powered automobiles, the effect caused by the anticipated change in the quantities of environmental pollutants emitted to the atmosphere dictates the need for more thorough investigations. The present report is prepared for the EPA to assess the environmental health impact as a result of conversion of light duty vehicles from gasoline-powered to diesel-powered engines. However, much of the information to date is on heavy duty diesel engines and might thus lead to biassed extrapolations.

Although diesel exhaust contains relatively low levels of CO, making it suitable for use in mining operations, it is considered by some to be potentially a major source of air pollution due to its propensity for emitting visible smoke and obnoxious odor. Particular attention, therefore, should be given to the latter parameters in order to evaluate their significance in promoting any deleterious health effects. The exhaust emissions from gasoline-powered automobiles, on the other hand, are steadily decreasing as a result of introduction of emission control devices. With the rates expected to decrease even further when the statutory emission standard becomes mandatory, the emissions from diesel-powered automobiles may become a significant factor in air pollution. Likewise, recent decreases in particulate emissions from stationary sources might make the effects of diesel emissions even more evident. It is, therefore, important to make a comparative study between diesel and gasoline exhaust with the objective of maintaining a comparable level (in terms of health effects) of environmental pollution.

Although the presently available literature contains abundant information in some aspects of exhaust emission rates from diesel- and gasoline-powered automobiles, there is a lack of data in other areas. In many cases, emission rate data determined with the objective of quantitating chemical species lack accompanying details about the engine operating conditions and vice versa. This makes the comparative study of emission rates between the two fuel-powered automobiles very difficult and/or impossible. The following section undertakes the task of presenting a comparative review of the physical and chemical significance of different exhaust emission parameters from diesel- and gasoline-powered automobiles, with particular emphasis on those with suspected injurious health effects.

3.0 Physical and Chemical Characteristics

The chemical compositions of diesel and gasoline emission have been broadly divided into two sections, one section detailing the particulate matter and the other vaporous emissions.

3.1 Particulates

The exhaust from both diesel and gasoline engines contain suspensions of microsize solid particles and liquid droplets in gas or vapor. It is, therefore, necessary to define which part of the exhaust can be considered as particulate matter. There is no general agreement on this subject. According to a commonly used definition anything other than condensed water that can be collected on Type A glass files filtering media at a temperature not to exceed 125°F (51.7°C) is considered as particulate matter. The choice of the type A filter is based on the fact that it removes 98% of the particles larger than 0.05 μm diameter (Sampson and Springer, 1973) from the gas or vapor phase. The selection of collection temperature of 125°F is on the basis of a compromise between minimization of moisture condensation and maximization of particulate collection. However, there are certain limitations to this definition. When the hot exhaust from the automobiles is discharged into the atmosphere, inhalation of particulate matter by humans occurs at ambient temperature after air dilution and associated cooling of the exhaust stream. Far more serious limitations of this definition may arise unless the retention efficiencies of the filtering media can be demonstrated to be high for particles of size ranges below 0.05 μm. Data regarding the efficiency of particulate collection from automobile exhaust are very limited. Therefore, until a better method is available, the particulate data developed by various authors should be interpreted in their proper perspective.

3.1.1 Physical Characteristics

The physical characteristics of the particulate from both diesel and gasoline exhaust are discussed individually.

Diesel Exhaust: X-ray spectroscopy shows soot to have a graphite structure with hexagonal basic carbon units linked into platelets giving a crystallite about 21 x 13A° containing 10 to 30 mole percent of hydrogen (Millington and French, 1966). The structure has a good resemblance to polybenzenoid substances, such as the polynuclear aromatic hydrocarbons (PNA's). The basic crystallite units agglomerate into spheres with a diameter range of 100-800A° (Vuk and Johnson, 1975). These agglomerates containing as few as one 100A° spherical particle or as many as 4000 spheres combine to form particles up to 30 μm diameter (Vuk et al., 1975). The other physical characteristics of the particulates as measured by different authors are presented in Table 3.1.

Table 3.1 shows that the particulate matter has very large surface area which make it a powerful adsorptive agent. The low still air settling velocity will make it remain airborne for a long period after generation.

It should be recognized that several parameters, such as, fuel composition, engine design and maintenance, operating conditions and emission control devices may influence the physical characteristics of the emitted particulates. The particle size is normally expressed in terms of either aerodynamic diameter or Mass Median Equivalent Diameter (MMED: diameter of an aerodynamically equivalent sphere of unit density). The effect of engine operating parameters, such as engine speed and load, on particle size was studied by Vuk and Johnson (1975). From their work these authors concluded

TABLE 3.1. PHYSICAL CHARACTERISTICS OF PARTICULATE FROM DIESEL EXHAUST

Parameter	Mass medium diameter (μm)	Particulate count no/cm^3	Surface area m^2/m^3	Settling velocity mm/hr.
Value	0.1 - 0.3	10^7	2	0.25 - 40
Reference	Vuk & Johnson, 1975, Dolan *et al.*, 1975, and Schreck, 1978	Frey & Corn, 1967	Frey & Corn, 1967	Frey & Corn, 1967

that the particle diameter decreases slightly with increasing engine load and temperature. Dolan et al. (1975), however, reported a shift in particle size shown as in Figure 3.1 from the smaller "nuclei mode" to the large "accumulation mode" with increasing engine load. The results of Laresgoiti et al. (1977) and Schreck (1978) do not show any significant variation in particle size either with engine speed or load. It can be concluded from these investigations that particle size may not be strongly dependent on engine operating conditions. Although no definite explanation can be offered, it can be conjectured that the discrepancies between the various results are due to the differences in engine design and fuel composition used in these experiments.

3.1.2 Gasoline Exhaust

The size of the particulate matter emitted from gasoline engine exhaust was studied by Mueller et al. (1962) and Moran and Manary (1970). Their findings were similar to that of Sampson and Springer (1973). The latter work is summarized below.

The particulate size from gasoline engine exhaust depends on the fuel composition. Unleaded fuel results in particulates of larger aerodynamic diameter. For leaded fuels, approximately 90 wt% of the emitted particulates are below 0.35 μm diameter and over 98 wt% below 10.0 μm diameter. In case of unleaded fuels, approximately 40 wt% of the total particulates are below 0.35 μm diameter and 88 wt% below 10.0 μm diameter. The particle size distributions of the emitted particulates from leaded and unleaded fuels was studied by Sampson and Springer (1973). They concluded that the weight of the smaller particles (<0.35 μm diameter) was much higher with leaded than with unleaded fuel.

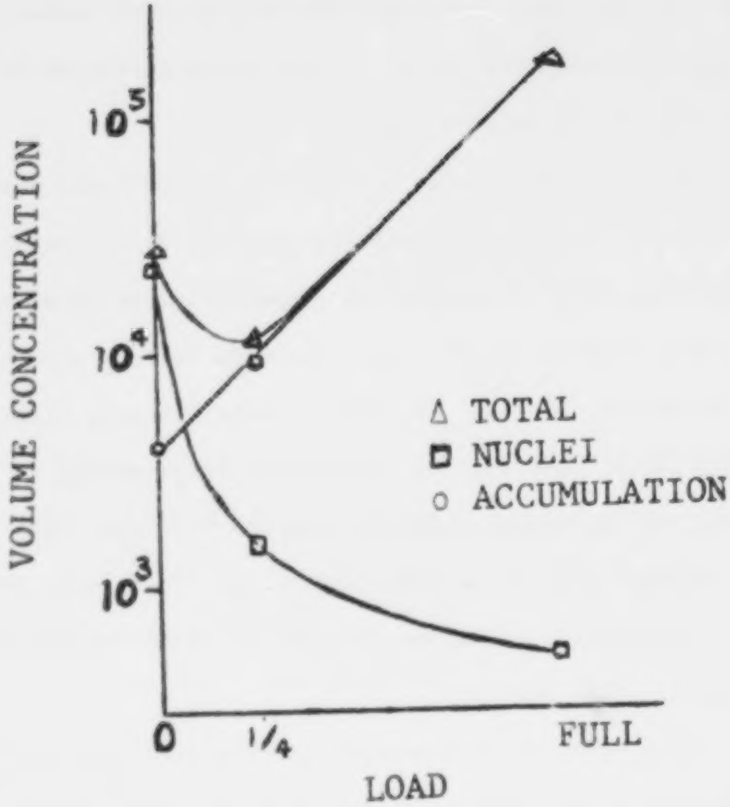

Figure 3.1. Variation in aerosol volume concentration with load for the two size components of diesel exhaust: the accumulation mode $0.08 \leq Dp \leq 2.0$ μm and the nuclei mode $Dp \leq 0.08$ μm (from Dolan et al., 1975). Dp: diameter of particles.

It has been estimated by Mueller (1970) that the MMED of the lead particles which can stay dispersed in the atmosphere fall in the range of 0.1 to 1.0 μm. Of the total lead particles emitted from leaded gasoline about 71-91% are respirable compared to 34-59% respirable particles in the total particulate matter in the exhaust (Mueller, 1970).

The particle size of the exhaust is similarly dependent on the fuel sulfur content. In gasoline vehicles equipped with a catalytic converter, the sulfate emission rate is substantial compared to the total particulate emission rate (see Section 3.1.6). More than 70% of the sulfate emitted by the vehicles may be in the form of H_2SO_4 with a geometric mean diameter of ∿0.02 μm (Wilson et al., 1977). The rest of the sulfate is primarily in the form of $(NH_4)_2SO_4$ and other refractory sulfates (Lee and Duffield, 1977). The mean diameter for ammonium sulfate aerosol is 0.07 μm. Therefore, the introduction of a catalytic converter to vehicles is bound to shift the emitted particulate diameter to smaller size ranges.

When sulfuric acid aerosol is exhausted into the atmosphere, most of the aerosol in the smaller nuclei mode undergoes growth into the larger accumulation mode in the size range of 0.1 to 1.0 μm. When this aerosol is inhaled, the high relative humidity in the pulmonary system causes the aerosol droplets to grow further in size. As a result of water vapor absorption the acid is also diluted. Thus Wilson et al. (1977) estimated that a 0.35 μm diameter droplet at 50% relative humidity would grow to a 1.0 μm in diameter at 99% relative humidity and the concentration would decrease from approximately 10.5 N to less than 0.5 N. But particles in the smaller nuclei mode will experience less growth and dilution due to the decrease in vapor pressure caused by the high curvature of small droplets. Both size ranges will deposit appreciably in the bronchi and alveolar regions. However, the nuclei mode will give greater total deposition and more deposition in the alveolar region.

The diameter of the emitted particles not only depends on fuel composition but also on engine operating conditions. Generally, it has been found that for leaded fuels cyclic operations yield larger particles than steady state operations (Habibi, 1970). The average size of the emitted lead particles, also, increase significantly with mileage accumulation from a MMED of 1.1 µm at 6350 average mileage to 4.7 µm at 21350 average mileage (Habibi, 1970).

The corresponding effects on the particle size for unleaded fuels has not yet been studied.

3.1.3 Emission Rate of Particulate Matter

The emission rates of particulate matter from both diesel- and gasoline-powered vehicles are presented in Table 3.2.

Several parameters affect the weight of particulates emitted from vehicles operated by both fuels. These parameters include fuel composition, engine design and maintenance, operating conditions, engine mileage, and the presence or absence of emission control devices.

In the case of diesel-powered vehicles, increase in fuel sulfur and aromaticity has been shown to increase particulate emissions (Braddock and Gabele, 1977). Similar increases in particulate emission with increase in fuel aromaticity has been observed by Ter Haar *et al.* (1972) in case of gasoline cars. The increase of particulate emission with increase in S-content of the

TABLE 3.2. PARTICULATE EMISSIONS FROM DIESEL- AND GASOLINE-POWERED PASSENGER CARS

Vehicle type	Engine displacement CID	Total particulates in FTP mode, mg/mile
Diesel vehicles:		
VW Rabbit[1]	90	294.0
Peugeot 504[2]	129	397.0
Mercedes 240D[3]	146	477.0
Mercedes 300D[3]	183	490.0
Oldsmobile 350[1]	350	924.0
Gasoline vehicles:		
With emission control		
VW Rabbit[1]	90	6.8
Oldsmobile 350[1]	350	9.1
Without emission control		
Leaded gasoline car[4]	a	246.0[b]
Unleaded gasoline car[4]	a	181.0[b]
Advanced unleaded gasoline car[5]	c	2.0

a. Various 1966-70 model cars.
b. Average of low and high mileage cars.
c. Data not available.
1. Ref. Springer and Baines, 1977
2. Ref. Braddock and Gabele, 1977
3. Ref. Springer and Stahman, 1977
4. Ref. TerHaar et al., 1972
5. EPA generated data cited in PEDCO Environ. Inc., 1978

fuel has been observed by Stara et al. (1974). Using a G.M. engine equipped with a pelletized catalyst, these authors have reported an increase of particulate emission from 9.6 mg/mile with 0.05% S indolene fuel to a value of 30.2 mg/mile with 0.10% S indolene fuel. The effect of fuel additives with non emission-controlled cars has been studied by Ter Haar et al. (1972). Addition of lead, carburator detergent, phosphorus and a commercial upper cylinder lubricant all caused increases in particulate emission.

The effect of engine size and operating modes on particulate emission rates has been studied by Springer and Stahman (1977) and Springer and Baines (1977). In the case of diesel-powered automobiles, the increase in particulate weight with the increase in engine size has been demonstrated by Springer and Stahman (1977). The effect of diesel engine operating parameters on the variation of particulate mass emission has been studied by Laresgoiti et al. (1977) with a Mercedes Benz Model 240D car and the effect is shown in Figure 3.2 and Figure 3.3.

From their work Laresgoiti et al. (1977) concluded that any change in engine performance parameters which affects a change in combustion temperature, combustion time, and fuel-to-air ratio in the combustion chamber will cause variation in the emitted particulates.

For gasoline-powered vehicles, moderate variations in the air-to-fuel ratio and spark timing has no significant effect on particulate emission rate (Ganley, 1973). However, an increase of the particulate weight by about 300% was observed as the engine speed and load was increased from 40 MPH to 70 MPH under road load conditions.

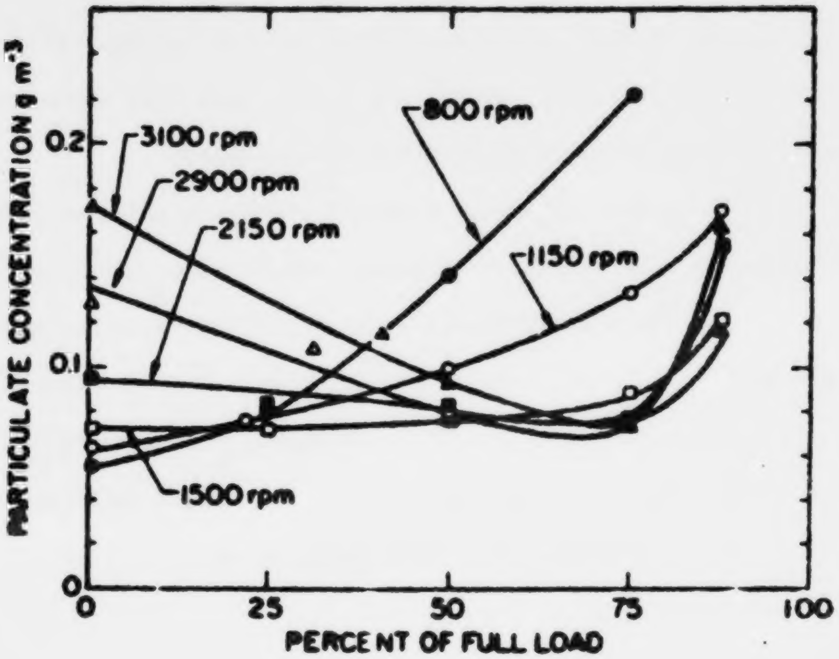

Figure 3.2. Exhaust particulate concentration as a function of engine speed. (Laresgoiti *et al.*, 977)

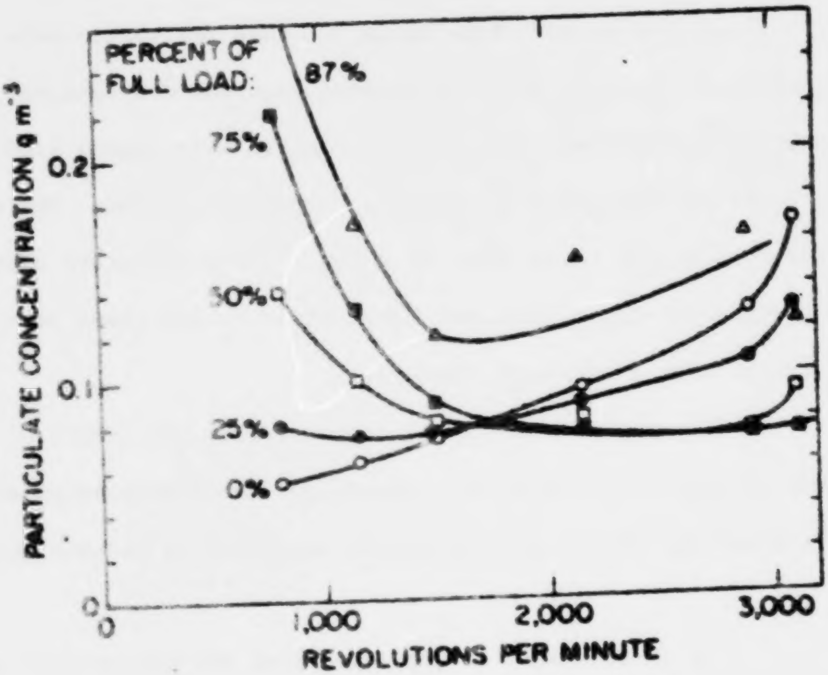

Figure 3.3. Exhaust particulate concentration as a function of engine load. (Laresgoiti et al., 1977)

The dependency of particulate emission rates for both diesel- and gasoline-powered automobiles on engine size and operating modes is shown in Table 3.3.

It can be seen from Table 3.3 that the particulate emission rate for both catalyst equipped gasoline-powered cars and non-catalyst equipped diesel-powered automobiles increases with increase in engine size and that the emission rates are dependent on the engine operating modes. The emission rates are higher during cold cycle than hot cycle. The particulate matter in exhaust from a non-catalyst equipped diesel is about 47 to 102 times more than gasoline engines fitted with a catalytic converter.

The use of a catalytic converter has the effect of increasing the amount of particulate from both diesel and gasoline-powered automobiles. Table 3.4 shows the effect of a catalytic converter on particulate emission rates.

It is necessary to emphasize that the particulate emission rate from non-catalyst cars operating with unleaded fuel has decreased substantially (see Table 3.2) with the introduction of advanced lean-burn engines, probably due to decreases in the sulfate emission rate. The primary reason for the increase in particulate matter with catalyst equipped cars is due to conversion of SO_2 to particulate sulfate (see Section 3.1.6).

The effect of different variables on the mode of particulate emission is qualitatively summarized in Table 3.5.

3.1.4 Chemical Composition

The chemical composition of particulate matter in both gasoline and diesel exhaust is complex. The characterization of diesel exhaust

TABLE 3.3. VARIATION OF PARTICULATE EMISSION RATES WITH CAR SIZE AND ENGINE OPERATING MODES[a]

Operating mode	Particulate emission rate, mg/km			
	Size of gasoline-powered car		Size of diesel-powered car	
	V.W. Rabbit 90 CID	Oldsmobile Cutlass 350 CID	V.W. Rabbit 90 CID	Oldsmobile Cutlass 350 CID
1975 Federal Test Procedure	3.84	5.63	182.0	573.0
Federal Test Procedure cold	4.95	8.38	202.0	628.0
Federal Test Procedure hot	3.01	3.55	165.0	523.0
Sulfur Emission Test	1.63	9.72	161.0	360.0
Fuel Economy Test	1.55	13.62	157.0	298.0

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a. Ref. Springer and Baines, 1977.

TABLE 3.4. EFFECT OF CATALYTIC CONVERTER ON PARTICULATE EMISSION RATES

Vehicle category	Particulate emission rate, mg/mile
Light-duty gasoline-powered vehicles:	
Catalyst[a]	6.0
Catalyst (excess air)[a]	15.0
Non-catalyst (lead fuel)[a]	250.0
Non-catalyst (unleaded fuel)[a]	2.0
Light-duty diesel-powered vehicles:	
Non-Catalyst[a]	500.0
Catalyst[b]	Some small decrease

a. EPA generated data cited in PED Co. Environ. Inc., 1978.
b. Ref. Seizinger, 1978

TABLE 3.5. EFFECT OF VARIABLES ON PARTICULATE EMISSION FROM GASOLINE- AND DIESEL-POWERED CARS

Variable	Emission Parameter	Effect		Reference
		Diesel	Gasoline	
Engine type:				
Indirect injection	Particulate mass	increase		EEA, Inc., 1978
Direct injection	Particulate mass	decrease		EEA, Inc., 1978
Turbocharging	Particulate mass	decrease		NIOSH, 1978
Engine displacement	Particulate mass	decrease with decrease in parameter	decrease with decrease in parameter	Springer & Baines, 1977
Emission control:				
Exhaust gas recirculation (EGR)	Particulate mass	increase		NIOSH, 1978
Catalyst	Particulate mass	increase	increase	Stewart et al., 1975 & Stara et al., 1974
Catalyst & water scrubber	Particulate mass	decrease		NIOSH, 1978
Thermal reactor	Particulate mass	decrease		NIOSH, 1978
Engine speed & load	Particulate size Particulate mass	no change increase with increase in variable	decrease increase with increase in variable	Lareegoiti et al., 1977 & Ganley, 1973
Engine maintenance:				
Engine deterioration	Particulate mass	increase with poor maintenance		NIOSH, 1978
Combustion chamber deposits	Particulate mass		increase with deposits	Gross, 1972
Fuel Composition				
S-content	Particulate mass	increase with increase in variable	increase with increase in variable	NIOSH, 1978 & Stara et al., 1974
Aromaticity	Particulate mass	increase with increase in variable	increase with increase in variable	Braddock & Gabele, 1977 & TerHaar et al., 1972
Fuel Additives				
Lead	Particulate mass		increase	Sampson & Springer, 1973
Halogen compounds	Particulate mass	increase	increase	Broome & Khan, 1971
Nitromethane	Particulate mass	decrease	decrease	Broome & Khan, 1971
Ba & Ni-additives	Particulate mass	decrease but may decrease engine life		Broome & Khan, 1971; Apostolescu et al., 1977
Methanol	Particulate mass	decrease		Broome & Khan, 1971
Water injected with fuel	Particulate mass	decrease		Greeves et al., 1977
Detergent	Particulate mass	Injector stays clean but no hard data on particulate emission	increase	NIOSH, 1978 & TerHaar, et al., 1972

particulates has begun only recently and the available information is limited. Complete combustion of fuel under perfect conditions should yield principally carbon dioxide and water. Because the combustion process in an actual engine is imperfect, several other products are produced. Both absolute and relative concentrations of combustion products are influenced by numerous factors. Some of the most prominent factors are: (1) air-to-fuel ratio, (2) ignition timing, (3) inlet mixture density, (4) combustion chamber geometry, (5) the variable parameters, such as speed, load, and engine temperatures, (6) fuel composition, and (7) presence of emission control devices.

The particulate matter from both diesel and gasoline exhaust contains a variety of products. Some of those which have been identified are: (1) unburned carbon, (2) unburned and partially burned hydrocarbons originating primarily from fuel and oil, (3) trace amounts of metals, (4) inorganic acids and their salts, namely, sulfates and nitrates, and (5) polycyclic aromatic hydrocarbons. The exhaust emissions from diesel and gasoline cars containing each of these categories of compound are discussed individually.

3.1.5 Trace Metals

The origin of metals in automobile emissions is from two distinct sources, namely, fuel and lube oil, and engine and exhaust system wear. When catalytic converters are used, the third possible source could be attrition products from the catalyst. However, no trace metals from the latter source have been reported. When leaded gasoline is used, obviously the predominant metal content in the exhaust is lead (Campbell and Dartnell, 1973). The lead emission rates from cars under cyclic operating conditions have been studied by Ter Haar et al. (1972); Habibi et al., (1970); Ninomiya et al. (1970).

Typical emission factors for metals cannot be derived from baseline characterization of auto exhaust by dynamometer tests as performed by EPA, since attempts are made to keep variability of additives, oils, and lubricants to a minimum. Emphasis is placed rather on the effect of emissions as a function of variations in operating conditions. The data cited in Table 3.6 reflect this because the different test cycles differed significantly in the average speed and variability of the operating mode. All the metal data, except the precatalyst Cu and Fe data, given in Table 3.6 were obtained by an X-ray fluorescence method. The precatalyst Fe and Cu data were obtained by emission spectrometry.

A comparison of the results given in Table 3.6 with the results obtained by Springer and Baines (1977) is interesting. With the exception of iron, the latter authors have failed to detect any other metals listed in Table 3.6 in all modes of operation with a 1976 Oldsmobile diesel Cutlass and a 1977 V.W. diesel Rabbit. Evidently, more research is needed in this field to establish the possible emissions of trace metals from both diesel- and gasoline-powered passenger cars.

3.1.6 Inorganic Acids and Their Salts

The sulfate emission rates for diesel- and gasoline-powered passenger cars are demonstrated in Table 3.7.

Comparison of the average values shows that the diesel has higher sulfur emission rates than non-catalyst gasoline cars. However, gasoline cars equipped with a catalyst have comparable sulfate emission rates to diesel cars. These emission rates are substantially lower for advanced non-catalyst and three-way catalyst gasoline-fueled vehicles. However, addition of

TABLE 3.6. EMISSION RATES OF SELECTED METALS FROM A VARIETY OF CARS UNDER DIFFERENT OPERATING CONDITIONS

Pre-catalyst cars[1] Metals	Pb (mg/mi)	Mn (mg/mi)	Cu (mg/mi)	Fe (mg/mi)	Ni (mg/mi)
FTP	40.5	N.A.[a]	N.A.	N.A.	N.D.[b]
FET	19.8	N.A.	N.A.	N.A.	N.D.
SET	20.0	N.A.	N.A.	N.A.	N.D.
60 mph Cruise[2]					
Catalyst cars (49-State Standard)[3]					
50 mph Cruise					
Mean	0.028	N.A.	0.016	0.029	N.A.
Median	0.015	N.A.	0	0.007	N.A.
Range	0-0.325	N.A.	0-0.293	0-0.341	N.A.
Catalyst cars (Calif. Standard)[4]					
FTP	0.03	N.D.	0.18	2.28	0.01
Dual catalyst[5]					
FTP	0.18	N.A.	0.12	0.56	4.12
FET	0.03	N.A.	0.01	0.03	0.27
SET	0.07	N.A.	0.02	0.07	0.50
Lean Burn Engine[4]					
FTP	6.69	0.05	0.06	1.17	N.D.
FET	3.76	0.12	0.08	0.07	N.D.
SET	7.00	0.07	0.08	0.15	N.D.
Stratified charge[4]					
FTP	N.D.	N.D.	N.D.	0.13	0.01
FET	N.D.	N.D.	N.D.	0.23	0.01
SET	0.12	0.01	N.D.	0.05	N.D.
Rotary[4]					
FTP	0.40	0.09	0.04	0.70	0.06
Diesel (#2 Natl. Avg. Fuel)[6]					
FTP	2.55	1.46	1.56	1.56	N.A.
FET	2.50	1.45	2.03	0.12	N.A.
SET	2.00	1.19	1.54	0.08	N.A.

[a] N.A.: not available

[b] N.D.: not detected

1 Unpublished data by R.L. Bradow cited in Lee and Duffield, 1977b.

2 Dow, 1970

3 DEC, 1976

4 Gabele et al., 1977

5 EPA, 1977

6 Braddock and Bradow, 1975

TABLE 3.7. SULFATE EMISSION RATES (mg/mile) FROM DIESEL VERSUS GASOLINE PASSENGER CARS

Vehicle type	Emission rate (mg/mile)
Diesel vehicles:	
V.W. Rabbit[1]	1.7
Peugeot 204D[1]	4.2
Peugeot 504[1]	6.5
Mercedes 220D[1] (compsun)	9.2
Mercedes 240D[2]	14.2
Mercedes 300D[2]	16.6
Oldsmobile 350[3]	21.0
Gasoline vehicles:	
Non-catalyst, no air pump	
Chevy, 1972[3]	0.2
Non-catalyst, air pump	
Granada, 1975[4]	0.7
Dodge, 1975[4]	2.7
Catalyst, pelletized, no air	
Average of 1975-76 cars[4]	10.1[a]
Catalyst, pelletized, air	
Average of 1975-76 cars[4]	12.8[a]
Catalyst, Monolith, no air	
Average of 1975-76 cars[4]	7.7
Catalyst, Monolith, air	
Average of 1975-76 cars[4]	33.5
Advanced non-catalyst	
Stratified charge[4]	1.9
Rotary, TRM reactor[4]	1.5
Lean burn[4]	1.0
Lean burn, TRM reactor[4]	6.3
Advanced catalyst	
3-way, F-I[4] (fuel injected)	1.5
3-way[4]	0.6[a]
Start cat.[3]	26.3
Dual cat.[4]	36.1
Lean burn oxd. cat.[4]	88.9
Sulfate trap[4]	4.7
Base metal cat.[4]	6.3

[a] One high value rejected in averaging the result.

[1] Ref. Springer and Baines, 1977

[2] Ref. Springer and Stahman, 1977

[3] Ref. [illegible] and Gabele, 1977

[4] Ref. Somers *et al.*, 1977

an air injected oxidation catalyst downstream of a three-way catalyst, or addition of an oxidation catalyst to a lean burn non-catalyst system, will result in higher sulfate emission rates typical of air pump equipped oxidation catalyst systems.

The sulfate emission rates for both classes of cars (diesel and gasoline) are dependent on four variables. These are: (1) vehicle design and engine displacement, (2) sulfur content of the fuel, (3) vehicle operation mode, and (4) engine mileage. The emission rates tend to increase proportionately to vehicle size, which is reasonable because this is the order of increasing fuel consumption. The effect of vehicle size and mode of operation on sulfate emission rates has been demonstrated by Springer and Baines (1977).

The national average for diesel and gasoline fuel supply sulfur content are 0.23% and 0.03%, respectively (Springer and Baines, 1977). Of the total fuel sulfur, 1-3% is converted to sulfate (Braddock and Gabele, 1977) in diesel engine operation. The conversion rate is similar in gasoline engines equipped with a catalytic converter (Springer and Baines, 1977). However, Wilson et al. (1977) have shown that the conversion rate can be as high as 13 ± 3% in gasoline cars fitted with a catalyst and air pump. Since diesel fuels contain about 8 times more sulfur than gasoline fuels, the increase of sulfate emission with increase in fuel sulfur content is pronounced in diesel vehicles. This is demonstrated in Table 3.8.

The effect of engine mileage on sulfate emission as noted by Lee and Duffield (1977) is evident in gasoline vehicles with catalytic converters. This is due to sulfate storage in the catalyst as shown by the reaction:

$$Al_2O_3 + 3\ SO_3 \rightleftharpoons Al_2\,(SO_4)_3$$

TABLE 3.8. SULFATE EMISSION RATES WITH THE VARIATION OF FUEL SULFUR CONTENT[a]

Fuel	Jet A	No. 1	No. 2[b]	No. 2D[c]	High-Sulfur No. 2D
Fuel % sulfur	0.04	0.13	0.23	0.29	0.49
Sulfate emission rates in SET mode, mg/mile	2.6	5.4	6.5	7.0	11.7

a. Ref. Braddock & Gabele, 1977

b. National average for No. 2 fuel oil.

c. With the exception of 26.5% aromatics content versus the minimum 27% specified, this fuel conforms with the No. 2D fuel specifications given in Federal Register for certification of light duty diesel engines.

On a fresh catalyst, much of the SO_3 that is formed is stored as $Al_2(SO_4)_3$. However, as the catalyst nears saturation, the reverse reaction becomes prominent. The equilibrium storage/release level of a catalyst is a function of catalyst temperature, catalyst space velocities, feed gas composition and time (Somers et al., 1977).

Although the rate of sulfate emission is shown in Table 3.7, nothing has been said about the form of sulfate emitted from automobiles, that is, how much of it is emitted in the free acid and salt state. This could be important in relation to the study of any possible effects on health. It is not known what types of sulfate are emitted by the diesel. In the case of gasoline cars, essentially all the sulfate is in sulfuric acid form (Lee and Duffield, 1977). A striking increase in the acidity of the particulate has been observed by Stara et al. (1974) when gasoline engine exhausts are treated with a catalytic converter. Comparison of the exhaust particulate acidity has shown that the acidity of catalyst-fitted engines is 65-260 times greater than engines with no catalytic converter (Stara et al., 1974).

The sulfuric acid mist emitted from vehicles is partly neutralized by ammonia to form $(NH_4)_2SO_4$ and NH_4HSO_4. A part of the acid reacts with other metallic elements or compounds in the atmosphere to form metal sulfates. However, it has been estimated by Wilson et al. (1977) that more than 70% of the sulfate emitted by the vehicles remains in the form of H_2SO_4 at 20 meters downwind from the point of emission.

The emission rates of hydrocyanic acid (HCN) as studied by Braddock and Gabele (1977) is discussed in Section 3.2.

Certainly, anions other than sulfate, such as nitrate and carbonate are emitted from automobile exhaust. Campbell and Dartnell (1972) have estimated that the nitrate ion from non-catalyst gasoline cars comprises 7.3% by weight of the total particulate. Stara et al. (1974) have reported nitrate emission rates from gasoline cars with and without a catalytic converter as 0.01 mg/mile and 0.04 mg/mile, respectively, at 15 MPH speed.

3.1.7 Elemental Carbon and Unburned and Partially Burned Fuel and Lubricant

The combustion of hydrocarbon fuels under "non-ideal" conditions involves complex processes. Some fuel passes the combustion zone unaltered, while some cracking processes in the ignition zone produce lower molecular weight compounds. Cracking processes out of contact with O_2 may produce elemental carbon. Some of the fuel is chemically rearranged by pyrolysis to produce fragments. Consequently, some new products can be formed by the interaction of various fragments of the fuel molecules. A part of the fuel undergoes oxidation producing partially oxidized products. Besides the fuel, lubricating oil is partially responsible for hydrocarbon emissions from automobiles.

Both absolute and relative concentrations of combustion products are influenced by numerous factors. Some of the most prominant factors are: (1) fuel-to-air ratio, (2) ignition timing, (3) inlet mixture density, (4) combustion chamber geometry, and (5) the variable parameters, such as speed, load and engine temperature. The fuel-to-air ratio influences the principal combustion products more than any other factors.

Although the literature has abundant data on the percent of carbon in the particulate matter from automobile emissions, the quantitative distribution of carbon into elemental and organically bound forms is not well established. One of the better methods for determining elemental carbon has been developed by Schreck et al. (1978). According to this method, the exhaust particulate matter is subjected to thermogravimetric analysis in a N_2 atmosphere from room temperature to 700°C. The particulate matter will start losing weight until all of the volatile organic matter has decomposed and/or volatilized and pure carbon particulates are left as residue. At 700°C, the atmosphere is changed to air. Oxidation of the remaining carbon particles will occur. When this process is complete, the remaining residue from the sample may be assumed to be due only to nonvolatile metallic compounds, chiefly as oxides. The residue from soxhlet extraction of particulate matter with benzene: ethanol (4:1) will also indicate the amount of unburned carbon particles and metallic compounds.

These methods were applied by Schreck et al. (1978) for the determination of elemental carbon in diesel exhaust. With a Peugeot 504D engine, these authors found that 40% of the particulate matter consisted of unburned elemental carbon and metallic compounds. Of the 40%, 31% was elemental carbon and 9% was metallic residue.

The value for percent elemental carbon from unleaded gasoline cars with emission controls was determined by Springer and Baines (1977) as 0.08%. It is evident from these results that the percent of elemental carbon from emission-controlled gasoline cars is very small compared to diesel automobiles. This is important to keep in mind since the PNA's usually have a tendency to remain adsorbed on the particulate phase, including the elemental carbon. Because of the absence of abundant particulate elemental carbon, the determination of PNA's in gasoline exhaust (with emission control) becomes especially difficult.

Determination of total hydrocarbons (THC) emissions from gasoline vehicles is usually performed by collecting the integrated cyclic emissions in a bag. Measurement of THC emissions from diesel motor vehicles is more difficult than with gasoline vehicles. The high boiling range fuels used in diesel preclude integration of the cyclic emissions in a bag since a major portion of the high molecular weight hydrocarbons are lost to the walls of the bag and other cool surfaces contacted. Thus, the cyclic emissions in diesel must be "real-time" integrated electronically.

Filtration of particulate matter from diesel emissions for hydrocarbon determinations poses several problems. First, the particulate adsorbed form must be distinguished from the free unadsorbed form. If the particulates are filtered at ambient temperature, the potential exists for adsorption of gaseous hydrocarbons on the carbon particles during their intimate contact on filtration. On the other hand, heating the filters could result in desorption of hydrocarbons which would remain particulate-bound under ambient conditions. This sensitivity of the gas phase-particulate phase partitioning of the organics to a variety of controllable sampling parameters was examined by Black and High (1978). The findings of their investigations are summarized below.

1. Instead of sample collection on hot (375°F) or cold (85°F) filters, a turbulent flow tunnel mixing system described in this work appears to adequately simulate the important parameters of short-term ambient dilution. The small variation of filter temperature under this sampling condition did not affect the particulate collection efficiency.

2. The probability of organic adsorption on the retained particulates on the filters was extremely low under this collection technique.

3. Isokinetic sampling is not important with diesel particulates under most conditions due to their very small aerodynamic size.

4. The distribution of organics between the gas and particulate phases is not sensitive to dilution ratio (dilution factor 8.7 to 17.9). There was indication of a small shift towards somewhat small particulate size at higher dilution rates.

5. The filtration media is also important as it was found that glass fiber media acted as sorption media for some gas phase organics. Best results were obtained with Teflon-coated glass fiber filter media (Pallflex Type T60 A20).

Determination of organic emissions by the above authors from diesel passenger cars driven under the cyclic patterns of the FTP showed that the hydrocarbons range from C_1 to about C_{40}. The hydrocarbons in the C_1 to C_{10} range result from the combustion process, that is, cracking from higher molecular weight organics. The C_1 to C_{10} organics are dominated by C_1, C_2, and C_3 hydrocarbons. The C_{10} to C_{40} organics are dominated by uncombusted fuel and lubricant and by partial combustion and rearrangement of these compounds. Some of the organics in this range (C_{10} to C_{40}) are particle bound and some are gas phase. Typically 15 to 20% of the THC from the vehicles are associated with the particles, but this can range to 40% with vehicles emitting significant levels of lubricant.

NMR determination of the solvent extracted portion of the particulate matter from a Peugeot 504D car showed that 45 wt% of the extract consisted of aromatic compounds with two or more rings (Schreck _et al_., 1978). Only 8% of the 45% consisted of three or more rings. Liquid exclusion chromatography (LEC) with styragel columns showed that the particle bound organics contained compounds with carbon numbers more than C_{93}. The origin of the higher carbon number compounds was probably lubricating oil (Schreck _et al_., 1978).

The values for the hydrocarbons from automobile emissions are normally reported as total hydrocarbon (THC) and volatile hydrocarbons. These values are given in later sections. Quantitative concentration values for the individual compounds in the particulate adsorbed hydrocarbons are not available. Various authors have attempted to identify these compounds. Boyer and Laitenen (1975) have detected hundreds of compounds in the molecular weight range 300 to 500 in gasoline automobile exhaust particulates by extraction and fractionation of the extract. The first fraction consisted of straight chain aliphatic hydrocarbons from $C_{16}H_{34}$ to $C_{33}H_{68}$. The second fraction contained branched chain aliphatics, unsaturated aliphatics and small ring compounds. The individual components in this fraction have not been identified. The third fraction consisted of PNA's. Because of the carcinogenic properties of some of these compounds the PNA's will be discussed individually (see Section 3.1.9). The fourth fraction contained mostly oxygenates. The individual components detected in this fraction by GC-MS are: 1) diethyl phthalate, 2) di-isobutyl phthalate, 3) di-n-butyl phthalate, 4) triphenyl phosphate, 5) di-n-octyl phthalate, 6) methyl triphenyl phosphate, 7) trimethyl triphenyl phosphate, 8) dimethyl triphenyl phosphate, 9) benzanthrone, 10) suspected β-capryophyllene, benzo[c]-cinnoline, benzoic acid, 2,6-di-tert-butyl hydroquinone or nonylphenol, and a number of other unidentifed compounds including oxygenated PNA's. The relative amounts of the classes of compounds has been estimated to be 50% saturated aliphatics, 5% PNA's and 30% oxygenated hydrocarbons (Boyer and Laitinen, 1975).

Organic compounds adsorbed on diesel exhaust particulates have been studied by Mentser and Sharkey (1977). The list of compounds detected by these authors by high resolution MS with diesel fuel oil No. 1 and No. 2 is as follows: 1) crotonaldehyde, 2) β-propriolactone, 3) unresolved 2-butanone, tetrahydrofuran, 4) pentane, 5) unresolved ethylformate, 2,3-epoxy-1-propanol,

methyl acetate, 6) carbon disulfide, 7) benzene, 8) pyridine, 9) cyclohexane, 10) cyclohexene, 11) methylacrylate, 12) 2-pentanone, 13) hexane, 14) unresolved dioxane, ethyl acetate, 15) toluene, 16) aniline, 17) phenol, 18) furfural, 19) furfuryl alcohol, 20) unresolved mesityl oxide, cyclohexanone, 21) methyl cyclohexane, 22) unresolved cyclohexanol, 2-hexanone, 23) unresolved ethyl acrylate, methyl methacrylate, 24) heptane, 25) styrene, 26) unresolved ethyl benzene, xylene, 27) unresolved monomethylaniline, O-toluidine, 28) cresol, 29) hydroquinone, 30) methylcyclohexanone, 31) allylglycidyl ether, 32) octane, 33) unresolved vinyl toluene, α-methyl styrene, 34) cumene, 35) isophorone, 36) p-tert-butyl toluene, 37) phenylglycidyl ether, 38) camphor, 39) phenyl ether, and 40) dinitro-o-cresol. The lower molecular weight compounds in the list have not been reported by other workers. Besides the above listed compounds, sulfur containing compounds, namely benzothiophenes and dibenzothiophenes have been detected in diesel exhaust (NIOSH, 1978).

The distribution of organic compounds in particulates changes in a consistent and characteristic manner as the speed and loading of the engines are increased from the idle to rated speed and full load. The composition profiles are not largely affected by the type of diesel engine or the type of fuel used (Mentser and Sharkey, 1977).

The above hydrocarbons in the presence of NO_x and light can be responsible for the formation of photochemical smog. Among the oxygenates, aldehydes are extremely reactive. Low molecular weight saturated ketones, alcohols, esters, and ethers are unreactive (Seizinger and Dimitriades, 1972). No reactivity data for heavier or unsaturated ketones, alcohols, ethers, and nitroalkanes have been reported. Seizinger and Dimitriades (1972) have suggested that the unsaturated oxygenates might possess significant reactivity.

3.1.8 Polycyclic Aromatic Compounds

This class of compounds is found in the automotive exhaust mostly in the particulate adsorbed phase. These compounds are discussed separately because of the demonstrated animal carcinogenic effects of some of these compounds.

One group of polycyclic aromatic compounds, the polycyclic aromatic hydrocarbons (PNA's), have been detected in both diesel and gasoline exhaust. These compounds originate from three sources: (1) PNA's present in original fuel, (2) synthesis from lower molecular weight hydrocarbons during fuel ignition, and (3) pyrolysis of lubricating oil. The mechanism of PNA formation in automotive engines has been demonstrated by Laity *et al*. (1973). PNA's apparently can exist in the quench zone at the surfaces of the combustion chamber. Some of these PNA's are vaporized from the walls or deposits during engine operation. Anything that increases the heat input to the combustion chamber walls, for example, advanced ignition timing, knock, use of hydrocarbon fuels, or high speed operation, leads to enhanced PNA emissions (Laity *et al*., 1973). From the examination of the soot particles obtained from a gasoline-powered passenger car and a diesel-powered omnibus, Lyons qualitatively detected a series of PNA compounds shown in Table 3.9.

Quantitative comparison of the PNA emission levels from diesel and gasoline exhaust requires that the vehicles at least be of similar duty category and operated under typical driving and fuel conditions. Although several publications have reported the levels of various PNA in the exhaust from gasoline- and diesel-powered vehicles, the results in most cases cannot be used for comparative purposes because either the engine, fuel, or driving

TABLE 3.9. PNA's DETECTED IN VARIOUS ATMOSPHERIC POLLUTANT SAMPLES[a]

Compound	Gasoline soot	Diesel soot	Atmospheric soot
Naphthalene	+[b]	-[c]	-
Acenaphthylene	+	+	+
Anthracene	+	+	+
Phenanthrene	-	+	-
Anthracene derivatives	+	+	+
Pyrene	+	+	+
Fluoranthene	+	+	+
Alkyl pyrene	+	-	-
Benz(a)anthracene	+	+	+
Chrysene	+	-	+
Benzo(e)pyrene	+	+	+
Perylene	+	+	+
Benzo(a)pyrene	+	+	+
Benzo(ghi)perylene	+	+	+
Benzo(b)fluoranthene	+	+	+
Anthranthrene	+	+	+
Tetracene	+	-	-
Coronene	+	+	+
Dibenz(a,h)anthracene	+	-	-
Dibenzo(a,l)pyrene	+	+	-
Benzo(k)fluoranthene	+	+	+
Pentaphene	+	+	-
Dibenzo(a,l)naphthacene	+	-	-
Dibenzo(a,h)pyrene	+	-	-
Dibenzo(a,e)pyrene	+	-	-
Dibenzo(b,pqr)perylene	+	-	-
Dibenzofluorene ?	-	+	+
Tribenzo(h,rst)pentaphene	+	-	-
Indeno-1,2,3-fluoranthene ?	-	+	-

a. Ref. Lyons, 1962
b. Detected in sample
c. Not detected in sample

mode were not comparable. The average PNA emissions from typical diesel vehicles run under the 13-mode federal cycle, and typical gasoline automobiles run under European and American city driving schedules are presented in Table 3.10. The data from older cars are included since current data on detailed PNA analysis are not available.

The fuels used in the tests given in Table 3.10 are as follows: Diesel-2D diesel fuel with 26% aromatics; American cars - typical regular grade gasoline fuels with 25% aromatics; European cars - a blend containing 47.7% aromatics and 52.3% paraffins. It should be pointed out that the diesel vehicles in Table 3.10 are the heavy duty variety. Due to unavailability of data from light duty vehicles, the heavy duty vehicle has been used for the purpose of comparison.

A large uncertainty in PNA levels from automobile exhaust can be expected. The discrepency between reported PNA values exists primarily because of difficulties in sample collection and analytical procedures, and the dependence of PNA emissions on engine operating conditions. This is reflected in Table 3.11.

Compounds other than those listed in Table 3.9 and Table 3.10 have also been detected in exhausts from automobiles operated on both types of fuel. For example, Grimmer (1977) has reported six PNA's, two of which are unknown compounds of molecular weight 300 and the rest are cyclopento[cd]-pyrene, methylenebenzo[a]pyrene, methylenebenzo[e]pyrene, and methylenebenzo-[ghi]perylene. Grimmer (1977) felt that this group of compounds accounted for the predominant part of the carcinogenic effect observed with gasoline engine exhaust extracts in mouse skin-painting studies.

TABLE 3.10. COMPARISON OF PNA EMISSION RATES FROM HEAVY DUTY DIESEL- AND GASOLINE-POWERED VEHICLES

Compounds	Emission rates, μg/gal fuel burned		
	Diesel vehicles[a]	Typical 6 cyclinder 1956-1962 American cars (gasoline)[b]	1970 European car (gasoline)[c]
Anthracene	N.D.[d]	41	1486
Phenanthrene	6410	176	N.R.[e]
Phenanthrene derivatives	8280	N.R.	N.R.
Fluoranthene	253	872	891
Pyrene	349	1145	2159
Benz(a)anthracene	35[f]	N.R.	123
Chrysene	5	N.R.	246
Benzo(j+k)fluoranthene	N.R.	N.R.	33
Benzo(a)pyrene	22	147	63
Benzo(e)pyrene	4	205	147
Indeno(1,2,3,-cd)pyrene	N.R.	N.R.	97
Benzo(ghi)perylene	7	649	423
Anthranthrene	N.R.	27	N.R.
Coronene	N.R.	256	197
Perylene	N.R.	12	N.R.

a. Ref. Spindt, 1974
b. Ref. Hangebrauck, 1967
c. Ref. Candeli *et al*., 1974
d. N.D.: not detected
e. N.R.: not reported
f. Ref. Spindt, 1977

TABLE 3.11. FREQUENCY OF OCCURRENCE OF PNA's IN DIESEL EXHAUST PARTICULATES [a]

Formula	Compound	Carcinogenicity[b]	Mol. wt.	Frequency of occurrence in 30 samples
$C_{18}H_{12}$	Chrysene	±	228.0936	28
	Benzo(c)phenanthrene	+++		
	Benz(a)anthracene	+		
$C_{20}H_{12}$	Benzo(a)pyrene	+++	252.0936	16
	Benzo(b)fluoranthene	++		
	Benzo(j)fluoranthene	++		
$C_{20}H_{14}$	Benz(j)aceanthrylene	++	254.1092	2
$C_{20}H_{16}$	7,12-Dimethylbenz(a)anthracene	++++	256.1248	1
$C_{21}H_{14}$	Dibenzo(a,g)fluorene	+	266.1092	2
$C_{20}H_{13}N$	Dibenzo(c,g)carbazole	+++	267.1045	1
$C_{22}H_{12}$	Indeno(1,2,3-cd)pyrene	+	276.0936	3
$C_{22}H_{14}$	Dibenz(a,h)anthracene	+++	278.1092	2
	Dibenz(a,j)anthracene	+		
	Dibenz(a,c)anthracene	+		

a. Ref. Menster & Sharkey, 1977

b. Carcinogenicity: ±, uncertain; +, carcinogenic; ++, +++, ++++, strongly carcinogenic, as per NAS notation.

In a recent publication Wang et al. (1978) have speculated on the presence of 6-nitrobenzo[a]pyrene in gasoline automobile exhaust. The Ames Salmonella typhimurium assay of this compound has shown that this compound is a direct-acting mutagen with activity comparable to benzo[a]pyrene (Wang et al., 1978). In fact, the formation of these direct-acting mutagens (nitro-BaP) upon exposure of PNA to gaseous pollutants in smog has been demonstrated by Pitts et al. (1978). In simulated atmospheres containing 1 ppm NO_2 and traces of HNO_3, direct-acting mutagens are readily formed from both BaP and perylene, a non-mutagen in the Ames reversion assay (Pitts et al., 1978). The nitration reaction produces 6-nitro, 1-nitro and 3-nitro-isomers of BaP and 3-nitro-isomers of perylene. These authors also suggest that the nitro-derivatives of PNA may eventually photooxidize to polycyclic quinone.

Primarily because of its carcinogenicity and frequency of occurrence, BaP has typically been measured as an indicator of PNA emission from automobile exhausts. Consequently, the bulk of available data is in terms of BaP, although the use of BaP data as an indicator of other PNA's is highly questionable.

Polynuclear aza heterocyclics is another class of compounds in automobile exhaust which can contribute to carcinogenic activity. Sawicki et al. (1965) determined the amounts of poly aza arenes in gasoline automobile exhaust which are summarized in Table 3.12. However, these data were generated with cars not equipped with a catalyst and may be subject to change.

So far, the emission levels of PNA have been discussed without any specific reference to the dependency of emissions on other parameters. In fact, the PNA emissions, like all other exhaust emissions, are dependent on a

TABLE 3.12. CONCENTRATION OF POLY AZA ARENES IN AUTOMOTIVE EXHAUST[a]

Compound	Conc. in μg per g exhaust particulate
Benz(h)quinoline	0.2
Benz(c)acridine	0.4
Indenoquinolines	0.9
Dibenz(a,j)acridine	< 0.2
Dibenz(a,h)acridine	< 0.2
Alkylbenz(c)acridines	< 0.2

a. Ref. Sawicki *et al.*, 1965

number of parameters. These are: (1) vehicle characteristics and engine design, (2) engine operation mode, (3) engine maintenance, (4) fuel composition, and (5) exhaust emission control system. The effect of each individual parameter is discussed in the following sections.

3.1.8.1 Dependency of PNA Emission on Vehicle Characteristics

Since the objective of this report is to consider emissions from light duty vehicles only, emissions from heavy duty vehicles will not be discussed. Even in light duty automobiles, PNA emissions may be dependent on the engine displacement capacity of the automobile. Comparing a number of 1956-1964 V-8 and V-6 engines, Hangebrauck *et al*. (1967) have failed to detect any statistically significant difference in PNA emission rates between the two engines operated with gasoline. A similar conclusion has been reached by Springer and Baines (1977) from the comparison of two catalytically-equipped cars, one with a V-8 and the other with an I-4 engine, and both powered with gasoline.

In the case of diesel engines, BaP emission rates and their dependency on engine type are shown in Table 3.13. It can be seen from Table 3.13 that BaP emission rates may not only depend on engine displacement but also on the engine type. The Peugeot 504D engine with larger engine displacement showed a lower BaP emission rate per mile. However, this may be due to the fact that the Peugeot 504D results may have been subjected to sampling error.

3.1.8.2 Dependency of PNA Emission on Engine Operation Mode

The dependency of PNA emission on engine speed and load for a gasoline- and diesel-powered vehicle is shown in Table 3.14 and Table 3.15.

TABLE 3.13. DEPENDENCY OF BaP EMISSION RATES WITH ENGINE TYPE

Vehicle type	Engine displacement, CID	BaP emission rate, μg/mile
Oldsmobile, V-8	350	7.3[a]
VW Rabbit, I-4	90	4.3[a]
Peugeot 504D	129	1.6[b]

a. Ref. Springer & Baines, 1977
b. Ref. Braddock & Gabele, 1977

The marked contrast between gasoline and diesel engine exhausts can be noted from Table 3.14 and Table 3.15. Increasing the engine load resulted in a rapid decrease in PNA emission in the former, and a marked increase in the latter. As the speed of the engine increased, the quantity of PNA emitted decreased for the gasoline-powered automobile. No uniform variation of PNA production with speed was noted for diesel emissions.

The PNA emission rates given in Table 3.14 and Table 3.15 are for older model gasoline cars and the characteristics of the diesel engine are not identified. With new model gasoline cars equipped with a catalytic converter, the PNA emission rates can be expected to be substantially lower (see Section 3.1.8.6). The BaP emission rates for diesel-powered passenger cars under various recently-developed cyclic modes of operations are shown in Table 3.16. Corresponding results for gasoline cars are not available. That the BaP emission rates in the FTP mode are higher than in SET and FET modes is obvious from Table 3.16.

3.1.8.3 Variation of PNA Emission with Engine Maintenance

Both deposits in the combustion chamber and fouling of the fuel injection system (improper fuel-to-air ratios) can dramatically increase PNA emissions in vehicular exhaust. With a gasoline-powered automobile having combustion chamber deposits, Gross (1972) has shown that the amount of BaP emission could be as much as 6.5 times greater than for clean engines. The dramatic effect of fuel-to-air ratio, which controls the efficiency of diesel engine operation, on the variation of PNA emission is shown in Table 3.17. It is evident from Table 3.17 that diesel engines with improper

TABLE 3.14. VARIATION OF PNA EMISSION RATES WITH INCREASING LOAD AND CONSTANT SPEED OF 1000 r.p.m.[a]

Engine load	Emission rate, μg/min. for PNA compound							
	Pyrene		Benzo(a)pyrene		Benzo(ghi)perylene		Anthranthrene	
	Gasoline	Diesel[b]	Gasoline	Diesel	Gasoline	Diesel	Gasoline	Diesel
0	439	137	61	146	177	22	102	0
1/4	59	267	0	465	45	42	17	43
1/2	26	536	1	772	5	124	0.3	223
3/4	17	1800	0	1320	3	640	0.3	472
full	21	2500	0	876	2	1265	0.3	469

a. Ref. Kotin *et al.*, 1955 & Kotin *et al.*, 1954

b. All the diesel results were run under inefficient fuel injection systems.

TABLE 3.15. VARIATION OF PNA EMISSION RATES WITH INCREASING SPEED AND NO LOAD[a]

r.p.m.	Emission rate, μg/min. for PNA compound							
	Pyrene		Benzo(a)pyrene		Benzo(ghi)perylene		Anthranthrene	
	Gasoline	Diesel[b]	Gasoline	Diesel	Gasoline	Diesel	Gasoline	Diesel
500	225	N.R.[c]	120	N.R.	235	N.R.	153	N.R.
1000	439	137	61	146	177	22	102	0
1200	N.R.	208	N.R.	9	N.R.	79	N.R.	4.3
1400	N.R.	188	N.R.	80	N.R.	0	N.R.	20
1500	507	N.R.	33	N.R.	60	N.R.	36	N.R.
2000	374	N.R.	40	N.R.	73	N.R.	27	N.R.
2500	346	N.R.	25	N.R.	70	N.R.	31	N.R.
3000	121	N.R.	13	N.R.	85	N.R.	14	N.R.

a. Ref. Kotin *et al.*, 1955 and Kotin *et al.*, 1954
b. All the diesel results were run under inefficient fuel injection system.
c. N.R.: not reported.

TABLE 3.16. BENZO(a)PYRENE EMISSION RATES UNDER VARIOUS MODES OF ENGINE OPERATION

Cycle	BaP emission rate, μg/mile	
	Oldsmobile V-8[a]	V.W. Rabbit I-4[a]
FTP	7.3	4.3
FET	3.1	2.4
SET	4.0	2.5

a. Springer and Baines, 1977

TABLE 3.17. DEPENDENCY OF BaP EMISSION UNDER DIFFERENT DIESEL ENGINE MAINTENANCE CONDITIONS[a]

Load	BaP Emission rate, μg/min.	
	Efficient condition[b]	Inefficient condition[c]
0	0	9
1/4	0	47
1/2	0	437
3/4	0	432
full	0	1706

a. Ref. Kotin *et al*., 1955

b. Clean fuel injection system and obtaining samples from completely warmed-up engine.

c. Fouling of the fuel injection system and/or engine deterioration.

TABLE 3.17. DEPENDENCY OF BaP EMISSION UNDER DIFFERENT DIESEL ENGINE MAINTENANCE CONDITIONS[a]

Load	BaP Emission rate, μg/min.	
	Efficient condition[b]	Inefficient condition[c]
0	0	9
1/4	0	47
1/2	0	437
3/4	0	432
full	0	1706

a. Ref. Kotin et al., 1955

b. Clean fuel injection system and obtaining samples from completely warmed-up engine.

c. Fouling of the fuel injection system and/or engine deterioration.

maintenance can be a significantly greater source of PNA pollution. However, well maintained diesel engines may preclude PNA emission into the atmosphere to a remarkable degree. Optimization of fuel-to-air ratio, except during the warm-up period, can make a diesel engine almost completely free from PNA emissions into the atmosphere.

3.1.8.4 Effect of Fuel Composition

Increased fuel aromaticity generally causes an increase in PNA emissions from gasoline engines (Griffing et al., 1971; Candeli et al., 1974). Gross (1972) has shown that an increase in fuel aromaticity from 11% to 46% causes an increase of 134% in PNA emissions from uncontrolled gasoline cars. Begeman and Colucci (1970) have demonstrated that the emission of BaP and benz[a]anthracene increased by 5 and 3.5 times, respectively, by increasing the BaP content of fuel from 1.1 ppm to 4 ppm. Analogous results have been obtained by Rinehart et al. (1970). According to Gross (1972) fuel rich in BaP enhances the emission of the same compounds, only if there are deposits in the combustion chamber of the engine. Stichting Concawe (1974), however, has contradicted this result and has shown that PNA emission is on the average 40% lower with fuels without PNA than with fuels containing 1.3 ppm of BaP at about the same level of aromaticity. Candeli et al. (1975) have attempted to resolve the problem but have been unable to ascertain whether the observed increase in BaP emission is due to an increase in fuel aromaticity or to an increase in fuel PNA content.

Tests with two gasoline cars by Gross (1972) have shown that fuels containing a high-boiling naphtha displayed increased PNA emissions compared to fuels without the naphtha but with the same fuel aromatics and PNA levels. In a third vehicle, the naphtha effect has been shown to be reversed.

The immediate effect of the presence of lead in gasoline on PNA emission has been examined in several laboratories. Begeman and Colucci (1970) have shown both small increases and small decreases in PNA emission for the presence of lead in Indolene fuel. Griffing et al. (1971), employing two different 1967 vehicles, have not found any effect of lead on BaP emissions. A similar conclusion has been reached by Gross (1972) from examination of later model gasoline cars.

3.1.8.5 Effect of Engine Mileage on PNA Emission

The effect of engine mileage on PNA emissions from gasoline cars is evident from Table 3.18.

Hoffman et al. (1965) have reported BaP emission rates at two levels of oil consumption for the same V-8 engine. BaP emission rates equivalent to 19 and 250 μg/mile have been determined for oil consumption of 1 quart per 1600 miles and 1 quart per 200 miles, respectively. The 13 times greater emission for the high-oil-consumption test suggests that the source of BaP might have been from burning of oil.

3.1.8.6 Effect of Exhaust Emission Control

Table 3.19 shows the effectiveness of engine modification and emission control devices on PNA emission rates.

TABLE 3.18. EFFECT OF GASOLINE ENGINE MILEAGE ON PNA EMISSION[a]

Car Mileage	PNA emissions, μg/mile						
	BaP	Pyrene	BeP	Perylene	B(ghi)P	Anthranthrene	Coronene
19000	5.6	81	9.5	0.28	26.0	2.3	9.6
26000	4.2	70	8.1	0.78	35.0	0.64	10.7
49000	3.9	27	8.6	0.57	14.3	0.3	4.1
58000	21.5	119	23.5	1.38	77.0	3.17	32.2

a. Ref. Hagenbrauck *et al.*, 1967

TABLE 3.19. EFFECT OF EXHAUST EMISSION CONTROL ON PNA EMISSION

Type of control	BaP emission rate, μg/gal fuel consumed	% Reduction
Gasoline:		
Uncontrolled (1956-64)	170[a]	-
Uncontrolled, 1966	70[b]	0
Engine modification, 1968	19-25[b]	∿ 70
Air-injected RAM thermal reactor, 1968	1.6[b]	∿ 98
Catalyst equipped, 1970	1.1[b]	∿ 98
Diesel:		
Catalyst treated		80-90[c]
Water scrubber		30[c]
Catalyst + water scrubber		80-90[c]

a. Hangebrauck et al., 1967
b. Gross, 1972
c. NIOSH, 1978

3.2 Volatile Emissions

Table 3.20 summarizes the regulated gaseous emissions data from diesel-powered passenger cars. The dependence of these emissions on engine class and operating modes is obvious from this table.

The corresponding values from gasoline vehicles is shown in Table 3.21.

To make comparison easy, the federal light-duty emission standards are presented in Table 3.22.

From the emission rates given in these tables it can be concluded that diesels (without emission controls) can be a higher source of hydrocarbon pollution than catalyst-equipped gasoline cars. The CO emission rates for both diesel and gasoline cars are about equal with gasoline cars emitting more CO in the FTP and less in the FET and SET modes than diesel cars. The NO_x emission rates for the gasoline cars, on the other hand, are higher than for diesel cars in all modes of cyclic operation.

The individual hydrocarbon emission rates for diesel-powered passenger cars are given in Table 3.23. The corresponding values for gasoline cars are presented in Table 3.24. With the exception of methane and toluene, individual hydrocarbon emission levels are higher for diesel than for gasoline cars.

The emission rates of carbonyl compounds for diesel cars are shown in Table 3.25. The emission rates for carbonyl compounds for gasoline cars are shown in Table 3.26. It is evident from these tables that with the exception of crotonaldehyde, diesel cars emit more carbonyl compounds than catalyst-equipped gasoline cars. That the emission rates for aromatic aldehydes increase with fuel aromaticity is shown in Table 3.27 for cars without emission controls.

TABLE 3.20. GASEOUS EMISSIONS DATA FROM A VARIETY OF DIESEL CARS UNDER DIFFERENT ENGINE MODES

Vehicle	Engine displacement, lit	Emission rates, g/km								
		Hydrocarbons			CO			NO_x		
		FTP	FET	SET	FTP	FET	SET	FTP	FET	SET
Mercedes 220D[a]	2.20	0.11	0.08	0.06	0.81	0.48	0.55	0.65	0.56	0.57
Mercedes 240D[a]	2.4	0.18	0.06	0.06	0.60	0.38	0.45	0.79	0.80	0.78
Mercedes 300D[a]	3.0	0.10	0.06	0.08	0.53	0.36	0.39	1.07	0.99	0.98
Peugeot 204D[a]	1.36	0.69	0.48	0.54	1.06	0.57	0.71	0.42	0.34	0.33
Peugeot 504D[b]	2.11	0.29	0.07	0.12	0.88	0.37	0.51	1.63	1.20	1.33
Nissan 220C[c]	N.A.[e]	0.25	N.A.	N.A.	1.10	N.A.	N.A.	1.37	N.A.	N.A.
Oldsmobile[d]	5.74	0.47	0.21	0.27	1.24	0.63	0.79	0.70	0.59	0.59
V.W. Rabbit[d]	1.47	0.23	0.08	0.09	0.49	0.31	0.34	0.54	0.52	0.50

a. Ref. Springer and Stahman, 1977

b. Ref. Braddock and Gabele, 1977

c. Ref. EPA result cited in b

d. Ref. Springer and Baines, 1977

e. N.A.: Not available

TABLE 3.21. GASEOUS EMISSIONS DATA FROM A VARIETY OF GASOLINE CARS WITH AND WITHOUT CATALYST

Vehicle	Emission rates, g/km								
	Hydrocarbons			CO			NO_x		
	FTP	FET	SET	FTP	FET	SET	FTP	FET	SET
1977 Catalyst equipped Olds. Cutlass[a]	0.24	0.06	0.08	1.34	0.12	0.53	0.85	0.88	0.86
1977 Catalyst equipped V.W. Rabbit[a]	0.14	0.03	0.03	2.30	0.03	0.19	0.63	1.22	1.01
1970 Mercedes (no catalyst)[b]	1.66	N.A.[d]	N.A.	20.05	N.A.	N.A.	2.19	N.A.	N.A.
1968 Air-injected RAM thermal reactor vehicle[c]	0.04	N.A.	N.A.	2.60	N.A.	N.A.	1.18	N.A.	N.A.
1970 Catalyst equipped car[c]	0.25	N.A.	N.A.	4.96	N.A.	N.A.	0.43	N.A.	N.A.

a. Ref. Springer and Baines, 1977. This is a prototype automobile.

b. Ref. Springer, 1971

c. Ref. Gross, 1972

d. N.A.: not available

TABLE 3.22. FEDERAL LIGHT-DUTY EMISSION STANDARDS[a]

Year	Emission Standards, g/mile		
	Hydrocarbons	CO	NO_x
1977-79	1.5	15	2
1980	90% reduction from 1970 value	7	2
1981	90% reduction from 1970 value	90% reduction from 1970 value	1

[a] Ref. Public Law 95-95 issued Aug. 7, 1977.

TABLE 3.23. DETAILED HYDROCARBONS EMISSION RATES (mg/km) FOR DIESEL CARS DURING TRANSIENT CYCLES

Emission	Cycle	Mercedes 220D [a]	Mercedes 240D [a]	Mercedes 300D [a]	Peugeot 204D [a]	Cutlass[b]	V.W.[b] Rabbit
Methane	FTP	19.65	5.57	3.94	9.30	12.7	6.7
	SET	13.82	2.75	3.34	4.44	5.1	3.3
	FET	12.58	3.66	4.25	4.04	3.4	4.7
Ethylene	FTP	21.63	17.74	14.49	38.13	49.2	28.1
	SET	15.26	12.07	9.39	27.76	28.5	15.3
	FET	14.56	12.14	8.49	24.20	21.8	15.1
Acetylene	FTP	8.65	1.31	2.81	7.57	5.3	1.5
	SET	6.51	5.02	trace	5.08	2.6	1.2
	FET	6.07	8.94	3.50	4.77	1.9	1.7
Propylene	FTP	N.D.[c]	N.D.	N.D.	19.98	17.1	9.6
	SET	N.D.	N.D.	N.D.	10.93	8.9	4.5
	FET	N.D.	N.D.	N.D.	9.37	6.5	4.5
Ethane	FTP	N.A.[d]	N.A.	N.A.	N.A.	4.2	0.9
	SET	N.A.	N.A.	N.A.	N.A.	1.7	0.4
	FET	N.A.	N.A.	N.A.	N.A.	0.3	0.6
Propane	FTP	N.A.	N.A.	N.A.	N.A.	0.1	N.D.
	SET	N.A.	N.A.	N.A.	N.A.	N.D.	N.D.
	FET	N.A.	N.A.	N.A.	N.A.	N.D.	N.D.
Benzene	FTP	6.07	N.D.	2.51	N.D.	11.6	5.1
	SET	3.26	N.D.	N.D.	N.D.	6.4	2.9
	FET	4.99	N.D.	3.10	N.D.	4.9	3.2
Toluene	FTP	N.A.	N.A.	N.A.	N.A.	2.6	0.6
	SET	N.A.	N.A.	N.A.	N.A.	N.D.	1.6
	FET	N.A.	N.A.	N.A.	N.A.	0.9	N.D.

a. Ref. Springer and Stahman, 1977

b. Ref. Springer and Baines, 1977

c. N.D. not detected

d. N.A. not available

TABLE 3.24. DETAILED HYDOCARBON EMISSION RATES (mg/km) DURING TRANSIENT CYCLES OF GASOLINE CARS[a]

Emission	Cycle	Cutlass	V.W. Rabbit
Methane	FTP	29.5	33.0
	SET	24.2	17.4
	FET	18.4	14.6
Ethylene	FTP	18.2	15.2
	SET	6.3	1.1
	FET	2.9	2.0
Acetylene	FTP	1.1	2.6
	SET	N.D.[b]	N.D.
	FET	N.D.	N.D.
Propylene	FTP	8.2	4.0
	SET	N.D.	N.D.
	FET	N.D.	N.D.
Ethane	FTP	14.8	6.4
	SET	9.2	2.4
	FET	6.7	N.D.
Propane	FTP	N.D.	N.D.
	SET	N.D.	N.D.
	FET	N.D.	N.D.
Benzene	FTP	5.6	9.1
	SET	2.7	N.D.
	FET	0.8	0.4
Toluene	FTP	13.6	12.3
	SET	2.4	1.2
	FET	1.4	0.9

a. Ref. Springer and Baines, 1977

b. N.D.; not detected.

TABLE 3.25. EMISSION RATES (mg/km) FOR CARBONYL COMPOUNDS FROM DIESEL CARS DURING TRANSIENT CYCLES

Emission	Cycle	Mercedes 220D[a]	Mercedes 240D[a]	Mercedes 300D[a]	Peugeot 204D[a]	Cutlass[b]	V.W.[b] Rabbit
Formaldehyde	FTP	2.52	3.9	3.80	11.25	15.8	16.0
	SET	1.50	3.08	5.81	8.50	12.3	6.0
	FET	1.55	3.57	3.95	7.76	8.2	4.3
Acetaldehyde	FTP	1.00	1.13	1.11	4.28	6.5	5.0
	SET	N.D.[c]	0.55	N.D.	3.75	6.3	1.5
	FET	N.D.	1.19	N.D.	4.05	3.0	1.1
Acetone	FTP	8.37	1.47	9.41	3.01	35.7	2.6
	SET	10.15	2.99	6.53	1.58	5.1	1.0
	FET	4.31	2.46	1.45	4.61	3.3	2.7
Iso-butaldehyde	FTP	11.08	2.19	N.D.	8.75	18.5	16.0
	SET	15.96	3.58	N.D.	6.54	10.4	1.9
	FET	7.90	4.26	1.50	7.92	8.9	3.3
Crotonaldehyde	FTP	2.37	0.67	1.17	4.10	4.2	N.A.[d]
	SET	2.82	2.27	1.17	2.75	2.4	N.A.
	FET	1.75	1.51	N.D.	3.05	N.A.	N.A.
Hexanaldehyde	FTP	0.47	N.D.	N.D.	N.D.	N.A.	N.A.
	SET	1.81	0.13	N.D.	N.D.	N.A.	N.A.
	FET	1.55	0.67	0.45	N.D.	N.A.	N.A.
Benzaldehyde	FTP	N.D.	N.D.	N.D.	N.D.	1.8	2.7
	SET	N.D.	2.29	N.D.	N.D.	1.1	N.A.
	FET	N.D.	2.00	1.22	N.D.	N.A.	N.A.

a. Ref. Springer and Baines, 1977
b. Ref. Springer and Baines, 1977
c. N.D.: not detected.
d. N.A.: not available.

TABLE 3.26. DETAILED CARBONYL EMISSION RATES (mg/km) FOR GASOLINE CARS DURING TRANSIENT CYCLES[a]

Emission	Cycle	Cutlass	V.W. Rabbit
Formaldehyde	FTP	2.6	0.4
	SET	1.3	0.3
	FET	1.6	0.5
Acetaldehyde	FTP	0.4	N.A.[b]
	SET	N.A.	N.A.
	FET	N.A.	N.A.
Acetone	FTP	N.A.	N.A.
	SET	N.A.	N.A.
	FET	0.5	N.A.
Iso-butanaldehyde	FTP	3.8	2.6
	SET	1.8	2.1
	FET	6.5	6.4
Crotonaldehyde	FTP	7.2	32.1
	SET	0.6	4.3
	FET	1.2	3.7
Hexanaldehyde	FTP	N.A.	N.A.
	SET	N.A.	N.A.
	FET	N.A.	N.A.
Benzaldehyde	FTP	N.A.	2.7
	SET	5.3	2.2
	FET	0.8	1.0

a. Ref. Springer and Baines, 1977
b. N.A.: not available

TABLE 3.27. INCREASE IN AROMATIC ALDEHYDE EMISSION RATES FOR GASOLINE CARS WITH INCREASE IN FUEL AROMATICITY[a]

Fuel	Aromatics, mole %	Total aldehydes, ppm	Aromatic aldehyde, ppm
Unleaded Premium	46.6	65	13.6
Leaded Premium	30.8	72	6.1
Leaded Regular	27.3	69	5.8

a. Ref. Hinkamp et al., 1971

TABLE 3.28. PHENOL IN EXHAUST GAS[a]

Test vehicle	Phenol emission range, mg/gal		
	11% Aromatic fuels	28% Aromatic fuels	46% Aromatic fuels
1966, no emission control	110-179	279-435	653-776
1968, engine modification	78-165	314-406	535-691
1970, engine modication with spark retard	74-123	222-287	370-485
1968, air injected RAM thermal reactor	---	---	2
1970, catalyst equipped	---	---	2

[a] Ref. Gross (1972).

TABLE 3.29. SO_2 EMISSION RATE FROM VARIOUS CARS UNDER CYCLIC OPERATIONS[a]

Cycle	SO_2 Emission Rate mg/km			
	Mercedes 220D	Mercedes 240D	Mercedes 300D	Peugeot 204D
FTP	350	320	310	260
FET	270	270	320	210
SET	250	250	260	200

[a] Ref. Springer and Stahman, 1977.

TABLE 3.30. COMPARISON OF HCN AND COS EMISSIONS (mg/mile) FROM DIESEL AND GASOLINE CARS[a]

Emission	Cycle	Peugeot 504D	Honda CVCC	Lean-burn Chrysler	Dual Catalyst Hornet
HCN[b]	FTP	1.32 ± 0.31[c]	11.5 ± 2.0	4.44 ± 2.67	10.7 ± 1.6
	FET	0.63 ± 0.39	7.6 ± 0.5	NR[d]	7.2 ± 0.3
	SET	0.43 ± 0.05	NR	NR	5.2 ± 0.8
COS[e]	FTP	0.55 ± 0.15	0.06 ± 0.06	0.40 ± 0.35	NR
	FET	0.19 ± 0.22	0.51 ± 0.67	0.26 ± 0.16	NR
	SET	0.24 ± 0.14	NR	NR	NR

[a] Ref. Braddock & Gabele (1977).

[b] The diesel tests were run with national average diesel, and gasoline tests were run with unleaded gasoline.

[c] Means standard deviation from mean.

[d] The diesel tests were run with 0.46 weight% fuel sulfur, and gasoline tests were run with 0.03 weight% fuel sulfur.

Phenols have also been detected in exhaust gases from gasoline cars. Table 3.28 shows the phenol emission rates as the gasoline cars became more and more modified. It is evident from Table 3.28 that phenol emission increases with increase in fuel aromaticity. However, with the present catalyst equipped cars the effect may not be pronounced since the phenol emission rate is too low.

Sulfur dioxide emission rates from diesel cars are dependent on fuel sulfur content. As the fuel sulfur increases, SO_2 emission also increases (Braddock and Gabele, 1977). The SO_2 emission rate from a number of cars operating with national average fuel sulfur (0.23%) is shown in Table 3.29.

The emission rates of HCN and COS from both diesel and gasoline passenger cars are listed in Table 3.30.

3.3 Fuel Economy

For comparison purposes, Springer and Baines (1977) have used one large (Oldsmobile V-8) and one small car (V.W. Rabbit I-4) in each diesel and gasoline category. Their results are summarized as follows: fuel consumption (ℓ/100 Km) of the diesel Cutlass is consistently 26 to 29% lower than the gasoline car regardless of the driving cycle. In terms of fuel economy (mpg), the percent increase in miles per gallon for the diesel is 35 to 40% greater than the gasoline car. In the case of the Rabbit, the fuel consumption rates for the diesel are 42%, 39% and 33% lower for FTP, SET and FET tests, respectively, compared to the gasoline Rabbit. In terms of fuel economy, the corresponding percent increase amounts to 74%, 65% and 49%, respectively.

3.4 Smoke Results

The results of a diesel smoke test on the larger Oldsmobile Cutlass and smaller V.W. Rabbit car are presented in Table 3.31.

It should be noted that 3 to 4% opacity by the EPA smokemeter is at the limit of smoke visibility. Most of the time, both cars operated in this area with brief excursions during rapid throttle movement.

TABLE 3.31. PERCENT EXHAUST SMOKE OPACITY FOR TWO DIESEL CARS[a]

Condition	Cutlass		Rabbit	
	Cold start cycle	Hot start cycle	Cold start cycle	Hot start cycle
Start	16.3	7.8	72.9	27.4
Idle	4.4	4.1	4.5	0.4
First acceleration peak	21.4	7.5	7.4	3.0
Idle at 1255 sec.	5.2	4.3	0.5	0.3
Second acceleration peak	19.4	16.6	39.4	37.7

[a] Ref. Springer and Baines, 1977

3.5 Odor Rating

The odor rating by the Turk Kit method includes an overall "D" odor which is comprised of burnt-smoky "B", oily "O", aromatic "A", and pungent "P" qualities as determined by an odor panel. On an odor intensity series of one through four, the last is considered the strongest odor. The odor intensity determined by this method (odor panel) for a number of diesel cars under various engine modes of operation is given in detail by Springer and Stahman (1977) and Springer and Baines (1977). Since the objective of this report is to identify the chemical components, the odor rating based on this scale will not be discussed. The reader is referred to the previously mentioned investigations. Another system of odor rating called the Diesel Odorant Analytical System (DOAS) which expresses odorant as Total Intensity of Aroma (TIA) has been used by Springer and Baines (1977) for diesel cars.

At IIT Research Institute, Dravnieks and coworkers (Dravnieks et al., 1971; O'Donnell et al., 1970) have employed two high-resolution chromatographic columns for separation of diesel exhaust components in order to identify diesel odorants. Table 3.32 lists the odorants they determined quantitatively.

Another group which has been conducting odor-related research on diesel exhaust for a number of years is Arthur D. Little Co. Based on the results of their odor studies (Spicer et al., 1975), ADL investigators have identified a large number of aromatic compounds and their isomers which are listed in Table 3.33.

TABLE 3.32. EXHAUST CONCENTRATIONS OF SOME ODORANTS AS DETERMINED BY IITRI WITH HIGH-RESOLUTION CHROMATOGRAPHY[a]

Exhaust component	Concentration, ppm
Acetaldehyde	0.00003
n-Butanol	0.00017
Decane	0.00344
Methyl benzene	0.000009
C_5 Substituted benzene	0.000032
Allyl toluene	0.000037
Methylindan	0.000074
Benzaldehyde	0.000345
Naphthalene	0.00038
Methyl naphthalene	0.00034

[a] Ref. Dravnieks *et al*. (1971), O'Donnell *et al*. (1970). It is not clear from the original reference whether these estimates refer to actual exhaust concentrations or to exhaust which has been diluted 11 to 1.

TABLE 3.33. DIESEL EXHAUST ODORANTS IDENTIFIED BY ADL[a]

Compound	Composition	Odor note
Methylindan	$C_{10}H_{12}$	Irritation
Tetralin	$C_{10}H_{12}$	Rubbery sulfide
Dimethylindan	$C_{11}H_{14}$	Kerosene
Methyltetralin	$C_{11}H_{14}$	Naphthenate
Dimethyltetralin	$C_{12}H_{16}$	Kerosene
Trimethylindan	$C_{12}H_{16}$	Kerosene, irritation
Alkyltetralin	$C_{12}H_{16}$	Kerosene
Trimethyltetralin	$C_{13}H_{18}$	Irritation
Alkyltetralin	$C_{13}H_{18}$	Kerosene, pungent, acid
Alkylindene	$C_{12}H_{14}$	Heavy oil
Alkylindene	$C_{13}H_{16}$	Heavy oil
Monomethyl napthalene	$C_{11}H_{16}$	Mothball, irritation

[a] Ref. Spicer et al. (1975).

3.6 Noise

A summary of sound levels from diesel and gasoline cars under different driving conditions is shown in Table 3.34.

The driveby exterior rating for a diesel Cutlass has been found to be 5 dBA higher than for a gasoline Cutlass, while the Rabbit has shown the same dBA level under this driving condition. Interior noise levels are slightly higher with diesels of both makes during acceleration. The exterior driveby at a constant 48.3 Km/hr speed has shown slightly higher interior and exterior noise for the Cutlass, while the opposite is true for the gasoline Rabbit. Idle noise levels are noticeably higher with the diesel Rabbit.

TABLE 3.34. SUMMARY OF SOUND LEVEL MEASUREMENTS[a]

		Noise on dBA scale			
		Oldsmobile Cutlass		V.W. Rabbit	
Noise at	Driving mode	Gasoline	Diesel	Gasoline	Diesel
Exterior	Accel. driveby	68.8	73.8	71.0	71.5
Interior	Blower on	73.2	74.2	78.2	80.0
Interior	Blower off	68.8	70.5	76.5	79.5
Exterior	48.3 km/hr, driveby	58.8	61.2	60.5	58.5
Interior	Blower on	71.5	72.2	73.5	71.8
Interior	Blower off	60.5	64.0	70.5	68.0
Exterior	Idle	64.5	70.0	65.0	67.0
Interior	Blower on	71.5	71.0	69.5	69.5
Interior	Blower off	48.5	51.5	58.0	62.5

[a] Ref. Springer & Baines (1977).

3.7 Engine Modification and Antipollution Devices for Diesel Cars

Diesel powered cars discussed so far do not include automobiles equipped with antipollution devices. However, for diesel cars, the variation of combustion systems may result in different pollution characteristics and fuel economy. Table 3.35 shows qualitatively the characteristics of the two combustion systems without any other emission control system.

With the introduction of further emission control device(s), the pollution level can be further decreased in diesel cars. This is indicated in Table 3.36. Although the emission rates given in this table are for diesel vehicles run during mining operations, the results can be qualitatively applied towards diesel passenger cars. The effects of catalytic reactors, certain types of traps, and a combination of these, on diesel exhaust composition have been studied in detail by Marshall *et al*. (1978) and Seizinger (1978).

TABLE 3.35. FUEL ECONOMY AND EMISSION CHARACTERISTICS OF TWO DIESEL CARS WITH DIFFERENT COMBUSTION SYSTEMS[a]

Characteristics	Direct Injection	Indirect Injection
Fuel economy	Favorable	Less favorable
CO	Less favorable	Favorable
Hydrocarbons	Less favorable	Favorable
NO_x	Less favorable	Favorable
Aldehydes	Less favorable	Favorable
SO_2	Approximately same	Approximately same

a. Ref. NIOSH, 1978

TABLE 3.36. DIESEL EMISSION FACTORS WITH AND WITHOUT EMISSION CONTROL[a]

Pollutant	Emission level, grams/brake horsepower-hour			
	Untreated engine	Catalyst treated	Water scrubber	Catalyst and water scrubber
CO	0.6-2.7	0.6-0.3	0.6-2.7	0.6-0.3
Hydrocarbons	0.03-0.17	0.003-0.017	0.02-0.12	0.003-0.017
NO	1.25-4.1	1.25-3.5	1.25-4.1	1.25-3.5
NO_2	0.3-0.7	0.15-1.1	0.3-0.7	0.15-1.1
Carbon	0.17-0.67	0.17-0.67	0.12-0.47	0.08-0.33
Phenols	Trace	80-90% reduction	30% reduction	80-90% reduction
Aldehydes	0.02-0.2	0.005	0.01	0.005
SO_2	0.5	0.25	0.096	<0.09
H_2SO_4	—	0.37	--	<0.24
Trace metals	--	0.025 max	--	--
PNA	Trace	80-90% reduction	30% reduction	80-90% reduction
Odor		substantial reduction	--	--
Irritancy		--	some reduction	--
CO_2	510-600	no reduction	no reduction	no reduction
Noise, dBA	96-104	no reduction	no reduction	no reduction

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a. Ref. NIOSH, 1978

3.8 Effect of Irradiation of Automobile Exhaust

It has been long known that nitrogen oxides and hydrocarbons present in automobile exhaust can react photochemically in the presence of sunlight to produce 'photochemical smog.' The primary source of Los Angeles smog has been attributed to automobile emissions arising from evaporative fuel losses and exhaust discharges. Substantial research efforts have been devoted on this subject. No attempt has been made in this report to describe all these investigations. Instead, only a few investigations which demonstrate the alterations of major components as a result of photoreaction of exhaust emissions are presented below.

3.8.1 Photoreactivity of Gasoline Emissions

Light irradiation of gasoline emissions usually results in oxidation of products which are present in the original emissions. The reactivity criteria expressed in terms of rate of formation of O_3, NO_2, peroxyacetyl nitrate (PAN), peroxypropionyl nitrate, peroxybenzoyl nitrate (PBzN), formaldehyde and increase in eye irritation. In the case of gasoline cars, the effects of increased fuel aromaticity on photoreactivity of exhaust emissions have been studied by Heuss *et al.* (1974). This study is particularly important since unleaded gasoline used for catalytically equipped cars contains a higher percentage of aromatics in order to maintain the equivalent octane rating. The study by Heuss *et al.* (1974) has shown that the presence or absence of tetraethyl lead (TEL) in gasoline does not affect the photochemical reactivity of the exhaust hydrocarbons produced from the gasoline. Certain aromatics, when added to a low-aromatic gasoline, greatly increase the eye irritation and PBzN yield of the exhaust, although they do not increase other reactivity criteria.

The six aromatics tested by Heuss _et al_. (1974) have been ranked in the following order for their effect on eye irritation: isopropylbenzene > (_o_-xylene, _n_-propylbenzene, and ethylbenzene) > toluene > benzene. These authors concluded that the specific aromatics in gasoline, not the total, is the important factor affecting eye irritation.

Similar results on photochemical reactivity of gasoline exhaust in relation to increased fuel aromaticity have been estimated by Altshuller (1972). His results are summarized in Table 3.37.

3.8.2 Photoreactivity of Diesel Emissions

The photoreactivity of diesel emissions has been studied by EPA (1978). Their results show small but positive differences in the measured values of component concentrations between irradiated and non-irradiated diesel emissions. These differences exist between NO_x, SO_2, hydrocarbon and particulate matter in both atmospheres. Low molecular weight aliphatic hydrocarbons and solvent extractables do not show any significant differences. The EPA (1978) studies are summarized in Table 3.38.

TABLE 3.37. EFFECT OF PHOTOCHEMICAL REACTIVITY RESULTING FROM 10% INCREASE IN FUEL AROMATICITY[a]

Photochemical Reactivity	Probable Results
Ozone or oxidant	No increase overall and decrease for evaporative loss contribution
Peroxyacyl nitrate	No increase overall and decrease for evaporative loss contribution
Formaldehyde and other aldehydes	Decrease with increase in fuel aromaticity
Eye irritation	2 to 5% increase
Aerosol formation	10% increase
Plant damage	No change

a. Ref. Altschuller, 1972

TABLE 3.38. EFFECT OF IRRADIATION OF DIESEL EMISSIONS[a]

Component	Concentration in exposure chamber	
	Non-irradiated	Irradiated
CO_2%	0.252	0.255
CO, ppm	15.7	15.4
Total hydrocarbons, ppm C		
75°F	15.6	15.0
350°F	31.2	26.0
NO, ppm	5.85	4.94
NO_2, ppm	2.19	2.73
SO_2, ppm	2.13	1.91
O_3, ppm	--	<0.01
Total particulate, mg/m^3	6.32	6.83
Sulfate, mg/m^3	0.57	0.57

a. Ref. EPA, 1978

3.9 Research Gaps and Recommendations

The present review of the state-of-the-art knowledge on light duty vehicular emissions has detected the following areas of research gaps with regard to physical and chemical characterization.

3.9.1 Definition of Particulate Matter

There is no general agreement about the specific fraction of exhaust emissions which constitutes the particulate matter. Certainly, the nature and quantity of the particulate matter will depend on both the temperature of sample collection and the nature of the filtering medium. Therefore, a universally acceptable definition of particulate matter requires a standardization of these parameters.

3.9.2 Inadequate Particulate Sampling Procedure

This is perhaps one of the major reasons for both inter- and intralaboratory inconsistencies between the reported particulate matter concentrations from automobile exhausts. The turbulent flow tunnel mixing system described by Black and High (1978) seems promising. Although the filter paper and the cooling system for hot exhaust is optimized to accomplish consistent quantitative collection of particulates, hard data showing the actual collection efficiency are not available. Data by Spindt (1977) have shown that the collection efficiency for BaP can be less than 10%. In their experiments with gasoline exhausts, Springer and Baines (1977) point out the difficulty of collecting BaP present in particulate matter. Use of radioactive tracer to establish the recovery of particulate matter by the collection method used may be helpful.

The possibility of interactions among pollutants during collection (namely oxidants with PNA's) and during storage should be further investigated. This interaction may be responsible for the production of artifacts during these processes.

3.9.3 Better Storage Method

In cases where real time analyses are not possible, a better storage method for H_2SO_4 and other reactive components is needed.

3.9.4 Improvement of Analytical Methodology

For certain components where quantifications are hindered by interferences, namely, aldehydes, PNA's and phenols, better analytical methodologies need to be developed. There is a need for an instrument which will specifically measure NO_x concentration.

Better analytical procedures are also needed for the reasonable recovery of adsorbed components in the particulate matter during the solvent extraction procedure (perhaps ultrasonic extraction). Recovery during evaporative concentration of the extract can be improved by such well-established procedures as Kuderna-Danish evaporation.

3.9.5 Identification and Quantification of New Components

More research is required to identify and quantify suspected new carcinogenic components in the exhaust, namely, methylene-PNA's, (Grimmer, 1977) and nitro-PNA's, (Pitts et al., 1978), and hitherto undetected nitrosoamines. Fractionation of the extractables in the particulate matter into acid, base, and neutral fractions and the subsequent separation of each individual fraction may be helpful for this purpose. EPA has ongoing programs on the latter subject.

3.9.6 Analysis of Sulfates

A better method for the analysis of H_2SO_4 and other neutral sulfates from automobile exhaust is needed. Ion chromatography may be helpful for the detection of these and other ionic components. The time scale for neutralization of H_2SO_4 in ambient air has to be established.

3.9.7 Effect of NO and O_3 on Pollutant Formation

The effect of NO and O_3 on PNA and other compounds formed through free radical mechanisms should be investigated. Since NO and O_3 are well-known free radical quenchers, a relationship establishing NO and O_3 concentration and rate of PNA formation in automobile exhaust should be established.

3.9.8 Quantification of Different PNA Levels

More research effort should be directed towards establishing different PNA levels and measuring the effects of variables on PNA's emitted from light duty vehicles.

3.9.9 Uniformity in Data Reporting

EPA should establish a uniform method for data reporting. Comparison between various results sometimes becomes impossible because of the various units used to express the data.

3.9.10 Diesel Odor Characterization

Diesel odor characteristics as measured by DOAS and total "D" odor ratings by a human odor panel needs further evaluation because of their inherent inadequacies. Identification of odor components by chemical methods and rating odor on the basis of analytical quantification of a few representative odorants may be a solution to this problem.

3.9.11 Necessity for Using Additives

Use of a smoke suppresant for diesel exhaust is thought to be neither widespread nor essential. The use of MMT (C&EN News, 1978) or $Ce(thd)_4$

(Sievers and Sadlowski, 1978) to increase the anti-knock value for gasoline fuels needs careful examination. The best ways to control emission of pollutants from automobiles may be: (1) control of fuel parameters, (2) introduction of anti-pollution devices, and (3) engine modification.

3.9.12 Regulation of Pollutants

Finally, decisions have to be made regarding the necessity of regulation of some of the components in automobile emissions of possible interest in relation to health effects. The presently regulated gaseous emissions may be toxic and photochemically reactive, but are not principally mutagenic/carcinogenic. The regulation of vaporous components has unintentionally reduced the emission of some carcinogenic compounds (PNA's) in gasoline exhaust. Whether the presence of residual trace amounts of carcinogenic compounds poses any long term health hazards has to be assessed both by *in vitro* and long term *in vivo* biological studies.

References for Section 3.0

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4.0 Biological Effects

It has been known for many years that the exhaust emissions from both gasoline- and diesel-powered vehicles contain a variety of potentially toxic materials. Most prominent among these are several gaseous emissions: carbon monoxide, sulfur dioxide, oxides of nitrogen, aldehydes, and hydrocarbons. In addition, the presence of sulfates, metals, particulates, and polycyclic organic matter (POM) can be detected in varying amounts depending upon the type of fuel, engine load, and efficiency of operation (see Section 3.0).

The major public health concern regarding the use of diesel engines presently involves the particulate fraction of diesel exhaust. Recent analytical studies have shown that particulate emissions in diesel exhaust can be up to 82 times as much as in gasoline exhaust using paired vehicles (Springer and Baines, 1977). Emissions of carbon monoxide and volatile hydrocarbons are lower from diesel than from gasoline engines, although the use of an oxidation catalyst can substantially reduce most of these emissions from gasoline engines (Stara _et_ _al_., 1974; Lee _et_ _al_., 1976). There are several important reasons why increased exposure to particulates derived from diesel engines may constitute a potential health hazard (Schreck, 1978):

1) Carbonaceous particles from diesel exhaust are reportedly composed in part of high molecular weight polycyclic aromatic hydrocarbons.

2) These particles have high surface areas, theoretically enabling them to adsorb large quantities of gaseous exhaust products, most importantly the carcinogenic POM's such as benzo[a]pyrene.

3) The particles themselves may be degraded by atmospheric oxidation to yield lower molecular weight POM's which are potentially carcinogenic.

4) Diesel particulates are primarily in a size range (0.2-0.3 μm mean diameter) which would allow for deposition in the deep lung compartments, and possible retention in the lung.

The discussions presented in the following sections of this report summarize the major studies conducted thus far which indicate potential toxic reactions to diesel exhaust mixtures, particulate extracts, and fuel additives. Significant related studies using gasoline exhaust are included for comparison and clarification of toxicologic hazards resulting from combustion processes. Extensive health effects reviews have recently been published for many of the individual components of diesel exhaust such as carbon monoxide (National Academy of Sciences, 1977a), oxides of nitrogen (National Academy of Sciences, 1977b), particulates (National Academy of Sciences, 1977c), and polycyclic organic matter (Santodonato _et al_., 1978). Selected individual toxicants will be considered which make a particularly significant contribution to the overall toxic potential of the diesel exhaust mixture.

4.1 *In Vitro* Studies

4.1.1 Mutagenicity in Bacterial Systems

It has been shown recently that organic extracts of airborne particulate matter are mutagenic to histidine-requiring strains of *Salmonella typhimurium* (Teranishi *et al*., 1978; Dehnen *et al*., 1977). Because of the strong formal relationship between molecular events involved in mutagenesis and carcinogenesis (Miller, 1978), the demonstration of mutagenic activity for a substance is generally taken as strong presumptive evidence for the existence of carcinogenic activity as well. Therefore, it is believed that an investigation of the mutagenicity of foreign substances: (1) may be predictive of carcinogenic potential, (2) may be used to identify the most biologically active fractions of complex organic pollutants (e.g., diesel exhaust), and (3) may serve as an early warning of a possible threat to human health in cases where positive results are obtained.

The Ames *Salmonella* mutagenicity assay incorporating a mammalian microsomal preparation for activation of promutagens has received widespread use in environmental research. Studies sponsored by the U.S. Environmental Protection Agency have applied this assay to guide the fractionization of heavy-duty diesel exhaust by identifying biologically active components of the particulate fraction (Huisingh *et al*., 1978). Five histidine-requiring tester strains of *Salmonella typhimurium* were employed: TA 1535, TA 1537, TA 1538, TA 98, and TA 100. Strains TA 1537 and TA 1538 are reverted to histidine-independence by frameshift mutagens, while TA 1535 is reverted by mutagens causing base-pair substitutions. Strains TA 98 and TA 100, which contain a plasmid to increase sensitivity, respond to mutagens acting either by frameshift mutation or base-pair substitution.

Studies were carried out on fractions of a dichloromethane (DCM) extract of diesel exhaust particulate collected from two different engines on glass fiber filters. The DCM extract was divided into ether insoluble (INT), acidic (ACD), basic (BAS), and neutral (NUT) fractions; the NUT fraction being by far the largest and was further subdivided into paraffins (PRF), aromatics (ARM), a transitional (TRN) fraction, and a polar oxygenated (OXY) fraction (Figures 4.1 and 4.2).

Some mutagenic activity was demonstrated in the insoluble, basic, and acidic fractions extracted from both particulate samples. However, the neutral fraction showed most of the mutagenic activity. Within the neutral fraction, the paraffins subfraction was not mutagenic, whereas the transitional and oxygenated subfractions were highly active. Both direct-acting components and components requiring metabolic activation were apparent, although most of the mutagens were of the direct-acting frameshift type. These results are summarized in Figure 4.3. The observation of direct-acting mutagens is significant in that it excludes unsubstituted polycyclic hydrocarbons (e.g., benzo[a]pyrene) as causative agents, since they require metabolic activation for expression of mutagenic effects. Analysis of the specific components of the TRN and OXY subfractions was difficult, however, it was suggested that polar neutral compounds such as substituted polynuclear aromatics, phenols, ethers, and ketones were the major components.

Caution must be exercised before implicating polar neutral compounds in diesel particulate fractions as major health hazards to man. The human body, when confronted with airborne particulate pollutants, has no physiologic means to chemically fractionate these complex organic mixtures.

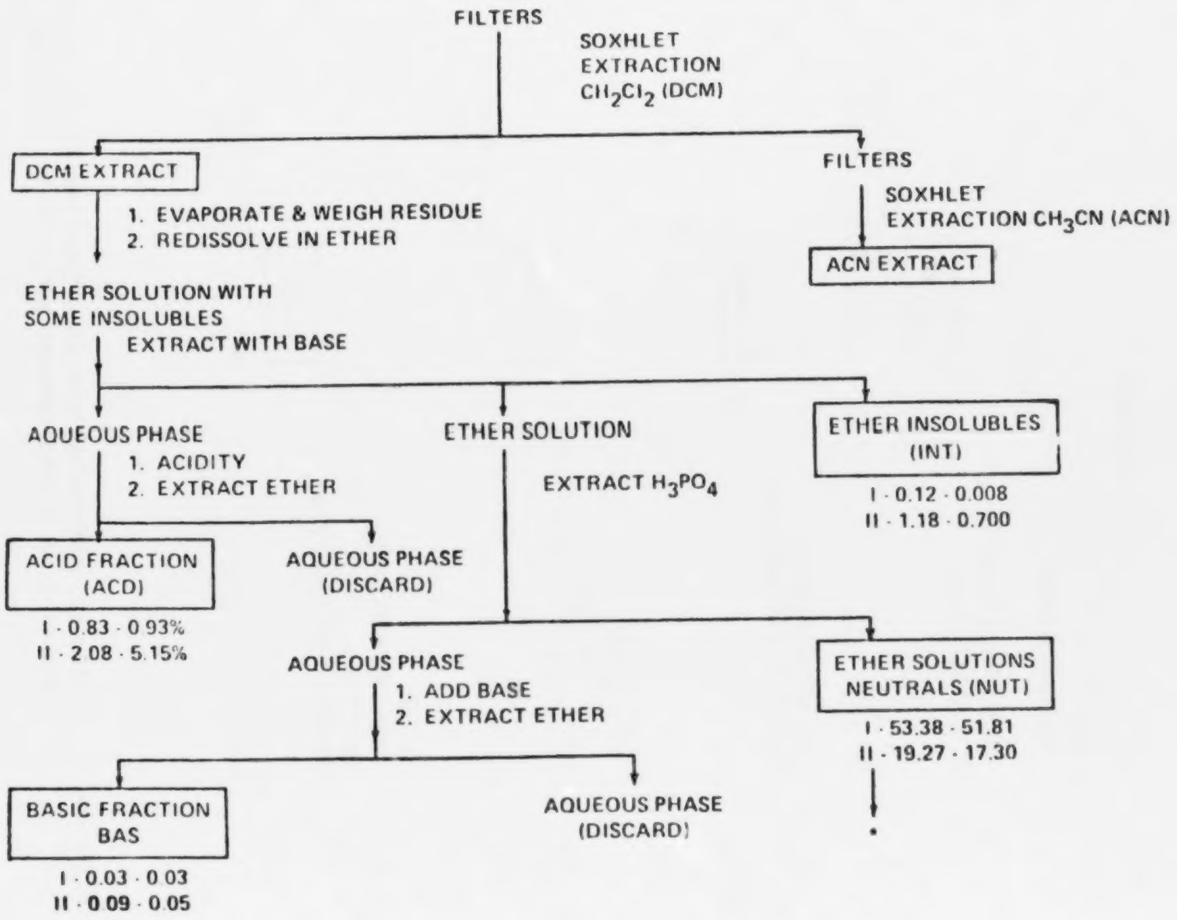

FILTERS
SOXHLET
EXTRACTION
CH_2Cl_2 (DCM)
DCM EXTRACT
1. EVAPORATE & WEIGH RESIDUE
2. REDISSOLVE IN ETHER
FILTERS
SOXHLET
EXTRACTION CH_3CN (ACN)
ACN EXTRACT
ETHER SOLUTION WITH
SOME INSOLUBLES
EXTRACT WITH BASE
AQUEOUS PHASE
1. ACIDITY
2. EXTRACT ETHER
ETHER SOLUTION
EXTRACT H_3PO_4
ETHER INSOLUBLES
(INT)
I · 0.12 · 0.008
II · 1.18 · 0.700
ACID FRACTION
(ACD)
I · 0.83 · 0.93%
II · 2.08 · 5.15%
AQUEOUS PHASE
(DISCARD)
AQUEOUS PHASE
1. ADD BASE
2. EXTRACT ETHER
ETHER SOLUTIONS
NEUTRALS (NUT)
I · 53.38 · 51.81
II · 19.27 · 17.30
BASIC FRACTION
BAS
I · 0.03 · 0.03
II · 0.09 · 0.05
AQUEOUS PHASE
(DISCARD)

Figure 4.1. Isolation and fractionation organics from diesel exhaust particulates (Huisingh et al., 1978)

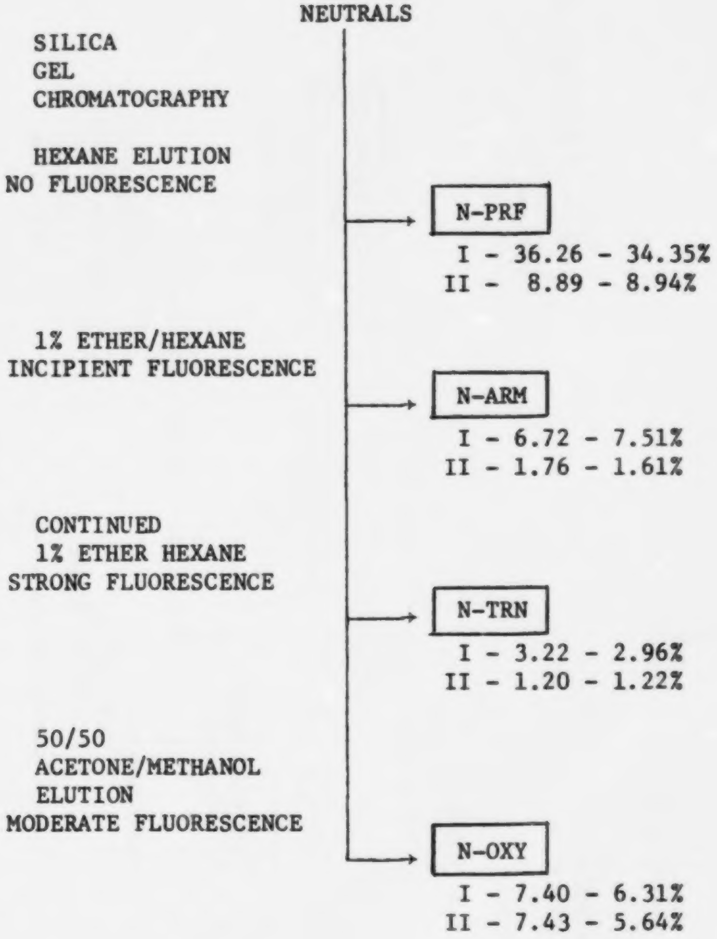
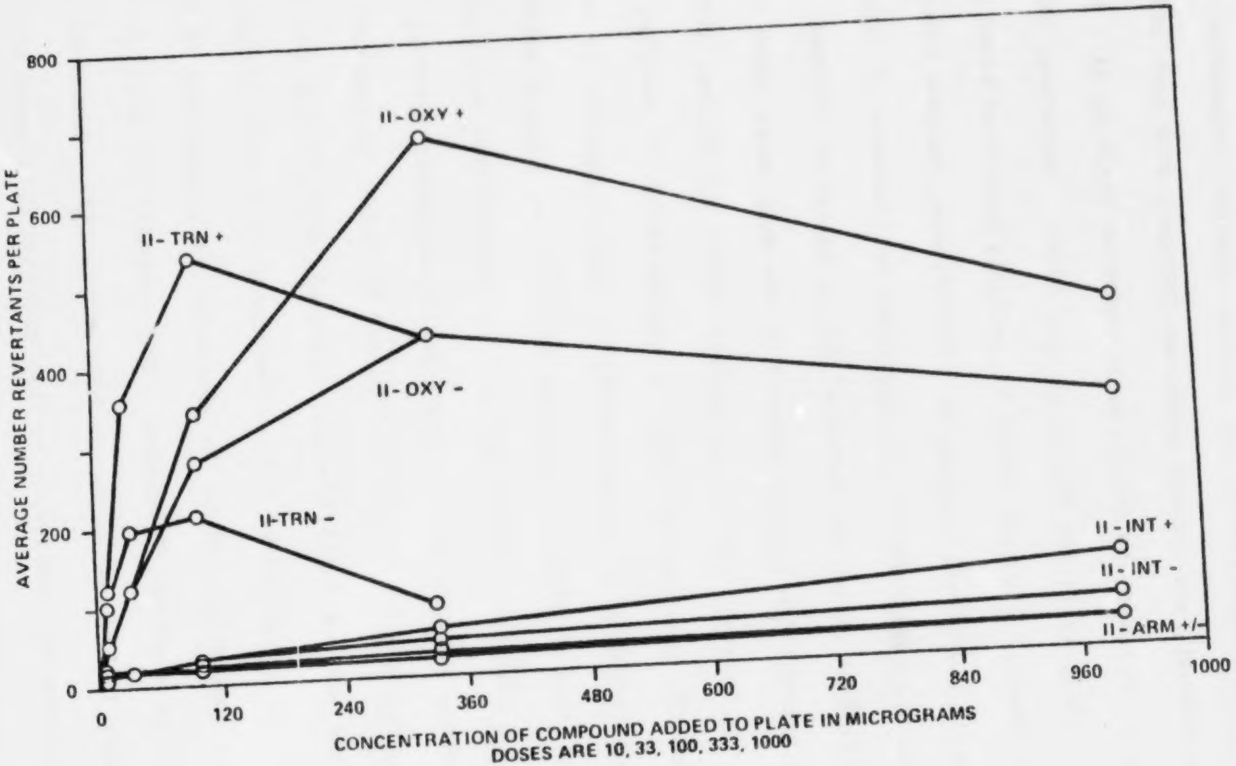

NEUTRALS
SILICA
GEL
CHROMATOGRAPHY
HEXANE ELUTION
NO FLUORESCENCE
N-PRF
I - 36.26 - 34.35%
II - 8.89 - 8.94%
1% ETHER/HEXANE
INCIPIENT FLUORESCENCE
N-ARM
I - 6.72 - 7.51%
II - 1.76 - 1.61%
CONTINUED
1% ETHER HEXANE
STRONG FLUORESCENCE
N-TRN
I - 3.22 - 2.96%
II - 1.20 - 1.22%
50/50
ACETONE/METHANOL
ELUTION
MODERATE FLUORESCENCE
N-OXY
I - 7.40 - 6.31%
II - 7.43 - 5.64%

Figure 4.2. Silica gel chromatography fractionation of the neutral organics from diesel exhaust particulate (Huisingh *et al*., 1978)



(+) = with metabolic activation; (-) = without metabolic activation

Figure 4.3. Comparison of the mutagenic response of various organic fractions from the 4-stroke cycle diesel truck exhaust particulate in Salmonella typhimurium strain TA 1538.

Target organs are thus simultaneously exposed to large numbers of environmental chemicals. It is well-established that various chemical components of polluted air, automobile exhaust, and tobacco smoke may interact with each other to either increase or decrease the carcinogenic response (Falk et al., 1964; Pfeiffer et al., 1973; 1977; Van Duuren et al., 1976). Therefore, the biological activity of chemical mixtures cannot be reliably predicted based on the specific actions of individual components. Furthermore, factors governing ease of absorption and biotransformation are important determinants of carcinogenic potency. These factors cannot be accounted for in bacterial systems. The apparent lack of correlation between potency in the Ames assay under certain conditions with carcinogenic potency in animals (Ashby and Styles, 1978a,b) further emphasizes the need for restraint in extrapolation of results.

The formation of direct-acting mutagenic compounds by combustion processes was confirmed in studies involving non-catalyst treated automobile exhaust (Wang et al., 1978). Acetone extracts of particulates collected from six different gasoline engines showed direct-acting mutagenic activity to Salmonella typhimurium strains TA 98, TA 100, and TA 1537. In contrast, unused motor oil and various fuels (leaded, unleaded, diesel) were not mutagenic. The postulated formation of nitro-substituted polycyclic aromatic hydrocarbons during combustion led to the synthesis and examination of 6-nitrobenzo[a]pyrene as a potential mutagen. This compound was found to be a direct-acting mutagen in strains TA 98, TA 100, and TA 1537. Mutagenic activity of 6-nitrobenzo[a]pyrene was comparable to that obtained with benzo[a]pyrene in the presence of a liver enzyme activating system. Since the mutagenic activity of particulate fractions of city air was correlated to the lead content of air, it was suggested that automobile emissions may be a primary source of direct-acting mutagens in the ambient atmosphere. However, nitro-substituted polycyclic compounds per se have not been monitored in the urban atmosphere.

Taken together, the results of bacterial mutagenicity assays on diesel and gasoline engine exhaust indicate that direct-acting mutagens are formed during combustion. The chemical identity of these substances is unknown, although substituted polycyclic aromatic hydrocarbons seem to be likely candidates in both cases. Presently there is no way to compare the mutagenic potency of gasoline versus diesel particulates since different collection and chemical fractionation schemes have been employed. Even if results were available from parallel Ames bioassays, the extrapolation of these data to support a health risk assessment would be limited to a qualitative judgement concerning cancer risk.

4.2 In Vivo Studies

4.2.1 Absorption, Metabolism, and Excretion

Exposure to diesel and gasoline engine exhaust occurs primarily by inhalation of gaseous and particulate emissions. Whereas highly water-soluble vapor phase organic emissions are generally absorbed across the moist surfaces of the upper respiratory tract, particulate material (depending upon size), water-insoluble compounds, and gases adsorbed to particulates may penetrate to the deeper lung compartments.

Studies on the deposition and retention of diesel particulate have considerable significance in light of the preponderance of this emission in comparison to that produced by the gasoline engine. Moreover, the likelihood that diesel particulate will contain adsorbed oxidants and POM presents a further dimension to the problem of potential toxic interaction with, or absorption across, the respiratory epithelium. In the absence of adsorbed substances with toxic potential, pure carbon particles as are formed during the diesel combustion process may not present a significant health threat.

Preliminary results are available concerning the physical characterization and clearance of diesel particulate from lungs of rats exposed to a 1:13 dilution of automotive diesel exhaust (Moore et al., 1978). The nature of the diesel particulate when collected on nucleopore membranes was examined by scanning electron microscopy. Particles smaller than 0.01 μm resembled spherical cotton balls while the larger respirable particulate had a flaky appearance, presumably due to the presence of adsorbed and condensed organic matter. Daily eight-hour exposures of rats to diesel exhaust lasting from one to 54 days produced a grey to black pigmentation of the lungs which varied in intensity with the duration of exposure. Black granular particulate material was observed histologically in the cytoplasm of the alveolar macrophages from all exposed animals. The diesel particulate could no longer be found in alveolar macrophages examined 28 days after a single eight-hour exposure.

Phagocytosis has been found to occur following the inhalation of diesel particulates. Examination of macrophages containing the phagocytosed particles has recently been conducted by transmission electron microscopy (Orthoefer et al., 1978). At 5000 times magnification, diesel particulate appears in the macrophage as an aggregation of small particles. Macrophages containing the particulate aggregates were also distinguished by the lack of primary vacuoles.

The quantitative aspects of particle clearance by macrophage ingestion are not well understood (NAS, 1977). It is generally believed that phagocytosed particles are transported by macrophages to the pharynx where they are subsequently swallowed. Thus, exposure to adsorbed chemicals may also occur via the gastrointestinal tract. In addition, it is suggested that clearance of macrophages from the lung may also lead to localization in various organs and tissues (Moore et al., 1978; Lauweryns and Baert, 1977). This process may have important implications for toxic effects in non-respiratory tissues. Insoluble particles, and presumably diesel particulate as well, can actually be partially digested by lysosomal hydrolases in the macrophage or remain trapped for the life of the cell. It is likely that the chemical nature of the diesel particulate will be the critical factor in determining its fate within the macrophage. Examination of the physical and chemical characteristics of diesel particulate, however, are complicated by the fact that particles and their agglomerates may be altered during the collection process prior to analysis.

The fate of POM adsorbed to diesel particulate can be inferred from studies involving BaP-coated carbon particles intratracheally instilled in mice (Creasia et al., 1976). When radiolabelled BaP was adsorbed to large carbon particles (15-30 μm) and instilled in the lungs, 50 percent of both the BaP and the carrier particles were cleared from the lungs in four to five days. Little carcinogen was released from the carbon particles in this case, and therefore, contact with the respiratory epithelium (and carcinogenicity) was low. With smaller carbon particles (0.5-1.0 μm), however, 50 percent particle clearance was not achieved until seven days after instillation. In this case, 15 percent of the adsorbed BaP was eluted from the particles and left free to react with the respiratory tissues. No measurements were made in this study of the phagocytic uptake of the particles by alveolar macrophages. In the complete absence of carrier particles, however, BaP was cleared from the lungs at 20 times the rate of adsorbed BaP.

In addition to mucociliary clearance and phagocytosis by alveolar macrophages, processes occurring in the lung which also determine the fate of adsorbed POM include metabolism by the respiratory tissues, and systemic absorption across the respiratory epithelium. It is known that BaP when administered intratracheally to rats appears in the body tissues with the same pattern of distribution as when given parenterally (Kotin et al., 1959). Similarly, Vainio and coworkers (1976) reported that unchanged BaP quickly appears in the perfusion fluid of isolated perfused rat lungs following intratracheal administration of a 200 nmole dose. The presence of particulate matter, however, can profoundly affect the rate and pathways of BaP metabolism in the isolated perfused lung (Warshawsky, 1978). When BaP and crude air particulate or ferric oxide were administered together, the rate of BaP metabolism was inhibited (Table 4.2). Pretreatment with particulate, on the other hand, caused a significant increase in the subsequent rate of BaP metabolism (Table 4.3). The enhancement of BaP metabolism by pretreatment with particulate apparently resulted from increased enzyme activity. Particulate-induced inhibition of the metabolism of co-administered BaP may have been due to the sequestering of adsorbed BaP in macrophages.

4.2.2 Acute Toxicity

4.2.2.1 Inhalation Exposure

The first comprehensive examination of the acute inhalation toxicity of diesel exhaust was conducted by Pattle and coworkers (1957). Their objective was to determine the principle toxic constituents of diesel exhaust generated under four conditions of engine operation: light load; moderate load; moderate load with "worn" fuel injector; light load with high fuel-to-air ratio. Mice, rabbits, and guinea pigs were severely exposed for five hours to the undiluted diesel exhaust. Under a light load, a highly acrid exhaust was produced which caused no mortality and minimal damage to the lungs. Exposures of greater duration (7 to 14 hours) under light load conditions produced nearly complete mortality in all species, accompanied by mild pathologic alterations in the trachea and lungs. Aldehydes (16 ppm) and oxides of nitrogen (46 ppm) were presumed to be the primary toxic agents in this case. Under moderate load conditions, a less irritating but more lethal exhaust was produced. Only slight alterations were seen in the trachea, but severe lung damage occurred,

Table 4.1 Influence of Particulates Administered to Isolated Perfused Lung on BaP Metabolism[*] (Warshawsky et al., 1978)

Pretreatment: IPL: No. of animals:	-- BaP 9	-- BaP + Fe_2O_2[a] 5	-- BaP + CAP[a] 5
Total rate of appearance of metabolites in blood (ng/hr/g lung ± SE)	256±38	165±51	156±42
Metabolic pattern in blood (%±SE)[b]			
7,8-Dihydrodiol	6.6±0.9	14.0±2.9[c]	19.1±4.4[d]
9,10-Dihydrodiol	15.4±4.0	20.4±1.9	28.3±7.9
4,5-Dihydrodiol	3.3±0.6	6.0±3.1	3.0±1.3
Monohydroxylated	9.7±1.1	5.4±1.6[e]	5.1±1.4[e]
Diones	10.6±1.8	5.6±1.5	5.2±2.6
Nonextractable	54.4±5.4	48.0±4.5	39.3±13.8

All three columns compared to each other. All metabolites separated by TLC.

[a] 1 mg/kg.
[b] Metabolite pattern values expressed as percent of total rate of appearance of metabolite in blood ±SE.
[c] $p = 0.05$.
[d] $p = 0.01$.
[e] $p = 0.1$ (by Student-Newman-Keuls test).
[*] abbreviations: IPL, isolated perfused lung; CAP, crude air particulate, BaP benzo[a]pyrene

Table 4.2. Influence of Particulate Pretreatment on BaP Metabolism*
(Modified from Warshawsky et al., 1978)

Pretreatment:	--	Fe_2O_3 IT[a]	CAP IT[a]	CAP IT[a]
IPL:	BaP	BaP	BaP	BaP+CAP
No. of animals	9	5	5	5
Total rate of appearance of metabolites in blood (ng/hr/g lung ± SE):	256±37	637±203[b]	830±100[c]	143±29[c]
Metabolite pattern in blood (% ± SE)[d]				
7,8-Dihydrodiol	6.6±0.9	13.3±2.3	18.2±5.6[b]	23.6±7.6
9,10-Dihydrodiol	15.4±4.0	26.3±5.6	32.6±4.3[b]	17.0±7.2
4,5-Dihydrodiol	3.3±0.6	2.9±2.0	0.9±0.5	1.9±0.7
Monohydroxylated	9.7±1.1	4.9±0.7[c]	3.4±0.6[c]	6.5±2.3
Diones	10.6±1.8	14.3±4.4	5.5±1.8	10.4±2.3
Nonextractable	54.4±5.4	37.7±5.8	39.4±8.0	40.6±6.6

All three columns compared to each other. All metabolites separated by TLC.

[a] 10 mg/kg, once/week x 5.

[b] p = 0.05 (by Student-Newman-Keuls test).

[c] p = 0.01.

[d] Metabolite pattern values expressed as percent of total rate of appearance of metabolite in blood ±SE.

* abbreviations: IPL, Isolated perfused lung; CAP, crud air particulate; BaP, benzo[a]pyrene

probably resulting from the high levels of nitrogen oxides (174-209 ppm) present in the exhaust. A rich combustion mixture produced the most lethal exhaust, killing all animals within five hours. The exhaust was high in aldehydes (154 ppm) and carbon monoxide (0.17%) and produced extreme irritation, only mild lung damage, but severe tracheal damage in rabbits and guinea pigs. Death was most likely due to carbon monoxide poisoning. Hydrocarbon levels were not determined in these experiments, and thus it is not known to what extent they may have contributed to the effects observed.

The acute effects of irradiated and nonirradiated gasoline engine exhaust in rats and hamsters were qualitatively similar to those produced by diesel exhaust as described above (Stara et al., 1974). After seven days of continuous inhalation exposure to gasoline exhaust (diluted at a ratio of 10:1), mortality in groups of infant rats reached 100% for irradiated exhaust and 77% for nonirradiated exhaust. The specific toxicant(s) responsible for the lethal effect was not determined, although death apparently did not result from carbon monoxide poisoning. In adult rats and hamsters exposed continuously for five days, various changes in lung morphology were noted, as well as vacuolar changes in hepatic parenchymal cells (irradiated and nonirradiated exhaust) and renal tubular cells (irradiated exhaust) of hamsters. In marked contrast to these results, gasoline exhaust from engines equipped with an oxidation catalyst produced virtually no mortality in infant rats or pathologic alterations in the tissues of adult rats and hamsters.

The importance of the particulate fraction in contributing to the acute effects of inhaled diesel exhaust was suggested in studies by

Battigelli and coworkers (1966). The effect of diluted diesel exhaust on tracheal clearance by mucociliary action was examined in rats exposed for cumulative periods of 4 to 100 hours. It has been previously established that noxious irritants and such gases as NO_2 and SO_2 can inhibit ciliary clearance and thereby render an organism more susceptible to respiratory infection and the actions of inhaled carcinogens (e.g., benzo[a]pyrene). As might be expected, inhalation of diluted diesel exhaust produced varying degrees of mucociliary inhibition which appeared to correlate with levels of NO_2 (1.9 - 15.0 ppm) and SO_2 (0.1 - 3.0 ppm) in the exhaust. Even more noteworthy, however, was the observation that the inhibition of clearance was markedly less in animals inhaling particle-free (filtered) exhaust. The particulate material was a complex mixture composed primarily of inorganic carbon (55-60%) and normal paraffins with chain lengths greater than four carbon atoms (35-38%). The authors concluded that the particulates in diesel exhaust may contribute directly to an adverse effect on host defenses, even in the absence of other gaseous emissions. The authors demonstrated that the inhibitory effect of a single exposure to diesel exhaust on mucociliary clearance was completely reversible within a few days.

Recent tests have now shown that female mice (CD-1, Charles River) inhaling diesel exhaust displayed enhanced mortality from respiratory infection by Streptococcus pyogenes (Campbell et al., 1978). Mice inhaled either irradiated or nonirradiated diesel exhaust, diluted with clean air at a ratio of 1:13, for a six hour period. Following the diesel exhaust exposure (within 1-2 hours), animals were briefly exposed to an aerosal of a broth culture of the test pathogen. Enhanced susceptibility to lethal infection was observed in all exhaust-treated groups, with those animals exposed to irradiated exhaust apparently being more severely affected. Enhanced susceptibility was not displayed when animals were challenged 22 hours after acute exposure. The contribution of NO_2 exposure to the observed increase in mortality is not known.

Both catalyst- and noncatalyst-treated gasoline engine exhaust can also enhance infective susceptibility in mice. Coffin and Blommer (1967) reported that mice exposed to diluted irradiated gasoline exhaust (no oxidation catalyst) for four hours experienced increased mortality from subsequent immediate exposure to streptococci. Exhaust diluted to yield carbon monoxide levels as low as 25 ppm and oxidant levels as low as .15 ppm was effective in increasing mortality to infectious pneumonia. In related studies using catalyst-treated gasoline engine exhaust (average dilution 1:14.1), irradiated exhaust caused a consistent and significantly greater susceptibility to infection than non-irradiated exhaust (Campbell _et al_., 1978). It was concluded, however, that relative to mortalities produced in clean air-treated controls, diesel exhaust was somewhat more effective than catalyst-treated gasoline engine exhaust in producing increased infection mortality.

Several studies have been concerned with the health effects of potential diesel fuel additives. Gutwein and coworkers (1972, 1974) conducted distribution and retention studies in rats acutely exposed to exhaust generated from diesel fuel containing a radiolabelled barium-based antismoke additive. Barium contained in the diesel exhaust (average concentration 1.39 mg/m^3) was transferred to the lungs, gastrointestinal tract, and bone during a 10 hour exposure. Whole body levels of barium reached 0.1 μg/g of tissue; clearance of barium from the lungs and gastrointestinal tract was rapid, whereas accumulation of the compound occurred in the bone. No observations were made for toxic symptoms resulting from absorption of barium or other components of the diesel exhaust.

4.2.3 Subacute Toxicity

4.2.3.1 Inhalation Exposure

A series of extensive studies has been initiated by the U.S. Environmental Protection Agency (EPA) regarding the effects of repeated inhalation of automotive diesel exhaust in animals. Preliminary results are available from several of these investigations where biological effects on selected parameters were measured at various intervals following initiation of exposure. Diesel exhaust used for these studies was generated with a Nissan CN6-33 engine coupled to a Chrysler Torque-flite automatic transmission. The engine was operated in a modified "California Cycle" using number 2 diesel fuel. A summary of exhaust component concentrations measured in the animal exposure chambers is presented in Table 4.3.

Moore and coworkers at EPA (1978a) exposed infant rats for 54 days (8 hours per day) to irradiated and non-irradiated diesel exhaust diluted at a ratio of 1:13. Clinical laboratory determinations were made during the study and selected animals were sacrificed for histologic examination of tissues at the termination of exposure. During the exposure period there was no mortality or adverse effects on body weight gain and general appearance of the animals. No significant differences in hematologic parameters or plasma electrolyte values could be shown between treated and control groups. However, reduced levels of alkaline phosphatase, serum glutamic-oxaloacetic transaminase (SGOT), and lactate dehydrogenase were evident in rats

TABLE 4.3. EXHAUST CONSTITUENTS AND CONDITIONS IN EXPOSURE CHAMBERS[a] (LEE *et al.*, 1978)

	Exposure Chamber Atmosphere		
Atmosphere Constituent or Condition	Purified Air (Control atmosphere)	Nonirradiated Diesel Exhaust	Irradiated Diesel Exhaust
Carbon monoxide (CO), ppm[b]	2.0	15.7	15.4
Total hydrocarbons (THC)[c], ppm (as carbon)	2.0	15.6	15.0
Nitric oxide (NO), ppm	0.11	5.85	4.94
Nitrogen dioxide, (NO_2), ppm	0.07	2.19	2.73
Sulfur dioxide (SO_2), ppm	NA[d]	2.13	1.91
Total suspended particulates (TSP), mg/m^3	NA	6.32	6.83
Sulfate $SO_4^=$ mg/m^3	0.0	0.57	0.57
Ozone ppm	--	--	<0.01
Carbon dioxide, (CO_2), mol %	0.040	0.252	0.255
Temperature, °C	24.0	23.7	24.1
Relative humidity, per cent	51.8	51.3	48.2

[a] Averages of weekly means

[b] ppm values are v/v

[c] By ambient-temperature probe flame ionization detector. Values using heated (350°F) probe are higher and may be approximated by multiplying by 1.9

[d] Data not yet available; values should be much lower than in exhaust chambers.

exposed to irradiated and non-irradiated diesel exhaust. Tissue damage generally results in increased serum levels of these enzymes, thus the toxicologic relevance of these observations is not known. Upon necropsy, minor histopathologic lesions of the respiratory tract were found. These included an accumulation of black pigmented alveolar macrophages throughout the lung. Black pigment found in the bronchial lymph nodes of one animal suggested clearance of particulate-laden macrophages. In the absence of any observed functional impairment, it is not possible to make a definitive statement regarding the severity of these pathologic changes. On the other hand, the presence of diesel particulate in the alveolar region and their clearance via the lymphatics indicates that adsorbed carcinogens are also delivered to these sites.

In further studies at EPA conducted with rats, the effect of a 28-day exposure to diesel exhaust on pulmonary function and arterial blood gases was evaluated (Pepelko et al., 1978). Groups of rats were exposed to diluted, irradiated and non-irradiated, diesel exhaust 20 hours per day, seven days per week. The slope of the static lung compliance curve and residual lung volume, both indicators of emphysematous change, were not affected by exposure to either irradiated or non-irradiated exhaust. Vital capacity and total lung capacity, which are non-specific indicators of change in pulmonary function, were both significantly increased in the group exposed to non-irradiated exhaust. This observation is consistent with previous results from studies with catalyst-treated automotive gasoline engine exhaust. It was not considered a serious effect in light of the duration of exposure. However, since these studies did not allow adequate time for the development of chronic lung disease, definitive conclusions cannot yet be drawn. Likewise, a lack of significant treatment effects on arterial blood gases may have reflected functional integrity only under conditions of short-term exposure. In addition, most clinical measures of pulmonary function are not ideally suited for the detection of very early lung damage.

More extensive studies on pulmonary function were conducted at EPA with cats exposed for 28 days to a 1:13 dilution of diesel exhaust (Pepelko _et al_., 1978a). Following completion of exposure, measurements were made of expiratory flow-volume curves, dynamic compliance, resistance, and pulmonary diffusing capacity. In addition, hematologic parameters were recorded and pathologic tissue evaluations conducted. No exposure-related physiologic effects were found other than a decrease in maximum expiratory flow rate at 10% of vital capacity; probably resulting from a slight increase in small airway resistance. This change can result from airway constriction under conditions such as smoking, chronic exposure to coal dust, or subclinical emphysema. However, pathologic examination of the respiratory tissues did not reveal emphysematous changes. The most prominent finding was the presence of focal alveolitis characterized by an accumulation of black pigmented clusters of one to 50 alveolar macrophages. These results support the conclusion that diesel exhaust particulate may penetrate to deep lung compartments and produce histopathologic changes, but do not allow for the prediction of possible chronic effects.

More serious physiologic and pathologic effects were found in infant guinea pigs exposed continuously (20 hours/day) to the diluted diesel exhaust for 28 or 56 days (Wiester _et al_., 1978). After four weeks of exposure to irradiated diesel exhaust, pulmonary flow resistance was substantially increased while dynamic compliance, minute volume, breathing rate, and tidal volume did not differ between exposed and control groups. The reason for the paradoxical increase only in pulmonary flow resistance was not evident. Exposure to either irradiated or nonirradiated exhaust caused increased lung weight to body weight ratios. In addition, guinea pigs exposed to irradiated exhaust displayed a slight but significant sinus bradycardia on electrocardiogram tracings. Other parameters of cardiac function and histopathology were normal. Thus the significance of the observed sinus bradycardia may have been more statistical than clinical. Animals sacrificed after 56 days of exposure revealed a characteristic focal alveolitis accompanied by pigmented macrophage accumulation, as was observed in cats and rats similarly exposed to diesel exhaust. The presence of black pigment in draining bronchial and carinal lymph nodes indicated a similar clearance mechanism for inhaled diesel particulate as was seen in the rat. Tissue response to diesel exhaust irritation was manifested by hypertrophy of goblet cells in the tracheobronchial tree; possible tissue damage was suggested by the presence of focal hyperplasia of alveolar lining cells, presumably Type II granular pneumocytes. There was no evidence of other changes such as squamous metaplasia, emphysema, peribronchitis, or peribronchiolitis.

It is apparent that the irritant effects produced by inhalation of diesel exhaust cannot be attributed solely to the action of carbonaceous particles, even though it is apparent that diesel particulate becomes widely distributed throughout the lung. At least some of the tissue damage produced by inhalation of diesel exhaust may be attributable to oxides of nitrogen, and particularly NO_2. On the other hand, water-soluble gases such as SO_2, as well as sulfate aerosols may contribute to functional disturbances (i.e., increased pulmonary flow resistance) but probably do not act in the alveolar region (NAS, 1977). The intervention of carrier particles having the proper size, however, can deliver irritant substances to the deepest lung compartments. Thus, diesel exhaust contains particulate which can adsorb irritant gases which are responsible for producing histopathologic damage that may lead to the development of emphysema. Moreover, the relative abundance of NO_x and particulates produced by diesel engines in comparison to gasoline engines suggests that the risk for chronic respiratory disease may be increased due to the greater potential for carrying adsorbed oxidants into the parenchymal region. The participation of adsorbed POM in eliciting irritation of the alveolar tissue is probably minimal (Santodonato et al., 1978).

Lee and coworkers at EPA (1978) have examined several biochemical parameters relevant to pulmonary fibrosis/emphysema and carcinogenesis that are influenced by exposure to diesel exhaust. In the rat, subchronic exposure to diesel exhaust produced a doubling in aryl hydrocarbon hydroxylase (AHH) activity, a measure of mixed-function oxidation, in the prostate and lung, and a 30% increase in the liver. Epoxide hydrase activity in these tissues was not increased by the diesel exposure. These microsomal enzyme systems are normally involved with detoxification of xenobiotics in conjunction with various P-450 type cytochromes. However, this system is also directly involved with the metabolism of carcinogenic polycyclic hydrocarbons to their active species (epoxides and diol-epoxides). Epoxide hydrase, also a microsomal enzyme, converts epoxides into vicinal glycols. Since some glycols are further metabolized by the mixed-function oxidases to form ultimate carcinogenic forms (i.e., diol-epoxides), this enzyme would likely affect both carcinogenesis and detoxification. Figure 4.4 presents a schematic representation of

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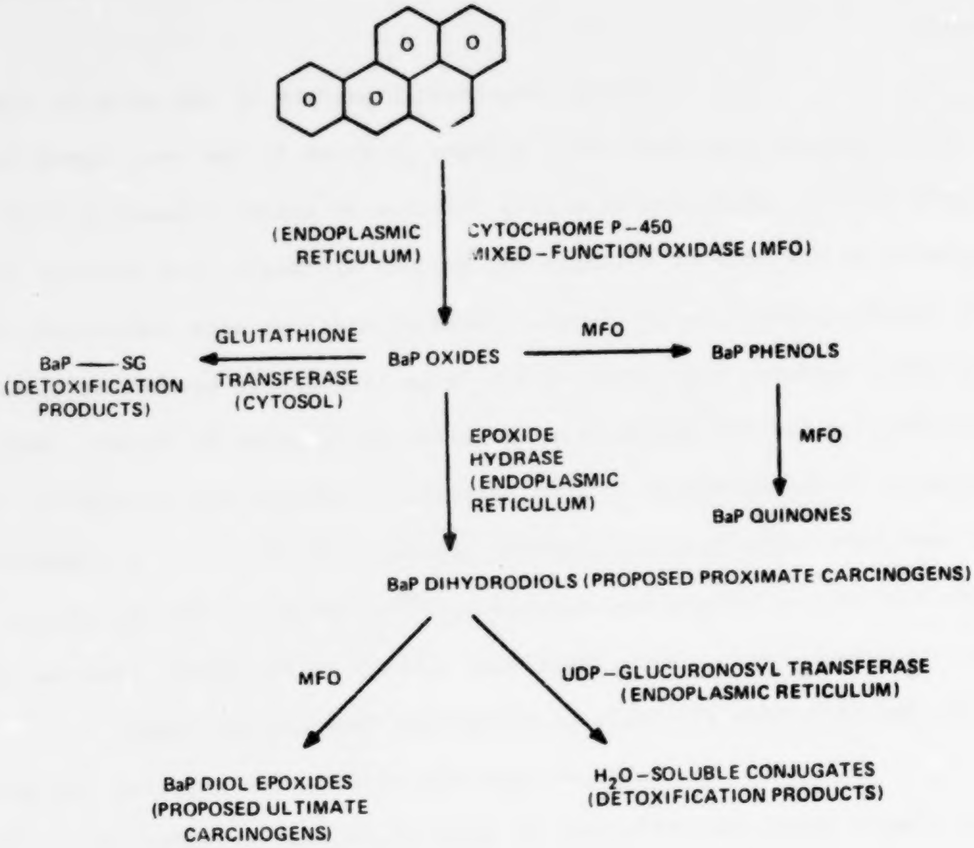
(ENDOPLASMIC RETICULUM)
CYTOCHROME P-450 MIXED-FUNCTION OXIDASE (MFO)
BaP — SG (DETOXIFICATION PRODUCTS)
GLUTATHIONE TRANSFERASE (CYTOSOL)
BaP OXIDES
MFO
BaP PHENOLS
EPOXIDE HYDRASE (ENDOPLASMIC RETICULUM)
MFO
BaP QUINONES
BaP DIHYDRODIOLS (PROPOSED PROXIMATE CARCINOGENS)
MFO
UDP-GLUCURONOSYL TRANSFERASE (ENDOPLASMIC RETICULUM)
BaP DIOL EPOXIDES (PROPOSED ULTIMATE CARCINOGENS)
H2O-SOLUBLE CONJUGATES (DETOXIFICATION PRODUCTS)

Figure 4.4. Enzymatic pathways involved in the activation and detoxification of BaP.

the various enzymes involved in activation and detoxification pathways for BaP, that is also representative of the known mechanisms of POM metabolism in general.

Further biochemical aspects of the subacute toxicity of diesel exhaust concerned early changes produced in the lung tissue (Lee et al., 1978). Inhalation of a 1:13 dilution of diesel exhaust by rats caused increases in the rate of collagen and protein synthesis, and enhanced prolylhydroxylase activity in the lungs. These alterations were indicative of fibrogenic changes, consistent with a large increase in connective tissue proliferation and continuous scar formation in response to injury. Such disturbances in the integrity of lung structure, although not necessarily linked, are important indicators of potential emphysema development. Accompanying these biochemical alterations were noticeable changes in the appearance of the diesel-exposed lungs. These lungs were rubbery to the touch, charcoal grey in color, and much more difficult to homogenize than control lungs.

Subacute exposure studies with gasoline engine exhaust have clearly shown the difference in toxicity between catalyst- and noncatalyst-treated exhaust (EPA, 1978). Infant guinea pigs exposed continuously for 35 days to diluted (1:10) catalyst-treated exhaust displayed reduced growth rate (0-20%), increased airway resistance (3-47%), and no change in lung compliance. Removal of the catalyst, however, resulted in growth rate reductions of 34-36%, increased airway resistance by 63-68%, and a significant decrease in lung compliance (35-39%). A severe increase in bronchial constriction was indicated in the non-catalyst group, which might represent a simple defense mechanism. Pathologic changes in the lungs of clean air controls and catalyst-treated exhaust groups included inflammatory lesions, focal thickening of the alveolar walls, and a focal pneumocytic hyperplasia. Similar, but more severe, changes were seen in the lungs of guinea pigs inhaling noncatalyst-treated exhaust. The changes observed in the control animals complicates the analysis of these results. Lactating female rats and their newborn offspring exposed to catalyst-treated gasoline exhaust for four and 12 weeks, respectively, displayed no treatment-related effects on mortality, body weight, hematology, or histopathology (EPA, 1978). The ability of an oxidation catalyst to reduce carbon monoxide levels in gasoline exhaust is credited with preventing the cardiac hypertrophy, and polycythemia which results from subchronic (4 week) exposure of rats to noncatalyst-treated exhaust. In addition, damage to the lung and/or kidney as shown by increased serum lactate dehydrogenase levels is also prevented by the catalyst.

Overall it is apparent that health-related benefits are derived from the use of an oxidation catalyst with gasoline engines. The prevention of significant subchronic effects by a catalytic converter can almost certainly be attributed to the substantial reductions which are realized in the emission of gaseous exhaust components. Subchronic exposure to diesel exhaust, on the other hand, produces damage which is apparently more severe than that produced by catalyst-treated gasoline exhaust, but somewhat less than that resulting from exposure with the catalyst removed.

Fuel additives have the potential to alter the chemical composition of engine emissions and possibly modify the biological effects produced by inhalation of exhaust. Moore and coworkers (1975) studied the biological effects of automotive emissions containing Mn particulate introduced by the fuel additive, methylcyclopentadienyl manganese tricarbonyl (MMT). MMT is used in unleaded gasoline as an antiknock additive, and is marketed as a smoke suppressant for diesel engines and stationary jet fuel power sources. Rats and hamsters were exposed eight hours per day for 56 consecutive days to gasoline engine exhaust (1:25 dilution) derived from fuel containing MMT at 0.25 g (as Mn) per gallon. Although increased tissue concentrations of Mn were produced by the exposure, no gross changes or histopathologic lesions could be attributed to the presence of MMT in the fuel. The primary lesion produced was a thickening of the cuboidal epithelium in the terminal bronchioles of the lung; an effect which was not considered particularly severe. Lesions did not become more severe with length of exposure, but occurred in 21% of the animals exposed to irradiated exhaust, 14% exposed to non-irradiated exhaust, and 60% of the clean air controls. It is noteworthy that the incidence of lesions in control animals costs doubts on the entire experiment.

4.2.3.2 Dermal Exposure

An early study has demonstrated that an organic extract of diesel exhaust particulates can produce severe systemic toxicity when applied to the skin of mice (Kotin _et_ _al_., 1955). Extracts of exhaust from a grossly inefficiently operating diesel engine when applied to the interscapular area of C57 black mice produced immediate tremors, followed by a reversible lethargy and loss of neuromuscular responses. Deaths began to result after about ten weeks of treatment (3 applications per week). Postmortem examination revealed a combined hepatotoxic and nephrotoxic effect. This was characterized by liver cord cell degeneration, and tubular degeneration in the lower nephron of the kidneys. The immediate cause of death in exhaust-treated mice was pneumonia, which was taken as evidence of decreased host resistance, although a mechanism was not postulated. When mice of the same strain were treated with extracts from gasoline engine exhaust (no oxidation catalyst) in the same manner as in the diesel studies, severe toxicity was not encountered (Kotin et al., 1954). The effect of the solvent employed in these experiments must be carefully considered, however.

4.2.3.3 Behavioral Effects

Behavioral alterations in rats have been produced by exposure to catalyst- and noncatalyst-treated gasoline engine exhaust (Cooper et al., 1977) as well as diluted diesel exhaust (Laurie et al., 1978). Concentrations of the various exhaust components are summarized in Table 4.4. Exposure of adult rats to diesel exhaust for 20 hours per day for six weeks caused a significant reduction in spontaneous locomotor activity measured at the end of the treatment period (Figure 4.5). In addition, decrements in forced activity performance resulted from the diesel exhaust exposure. During a four week recovery period, however, the difference in spontaneous locomotor activity between exhaust-treated and control rats was reduced.

In related studies, neonatal rats were exposed to diesel exhaust from day one after birth, 20 hours per day, for 17 days (Laurie et al., 1978). Measurements of surface righting and ear detachment taken after one or two days of exposure showed no differences from control values. Likewise, air righting, measured on days 14, 15, and 16, was not affected by the treatment. However, significant reductions were observed for both pivoting (measured on days 6 and 7) and eye opening (measured on days 14, 15, and 16) behavior when compared to controls. If data collected on female rats were considered alone, eye opening behavior was not significantly delayed.

It was concluded from these studies that spontaneous locomotor activity in adult rats was depressed further by exposure to diluted

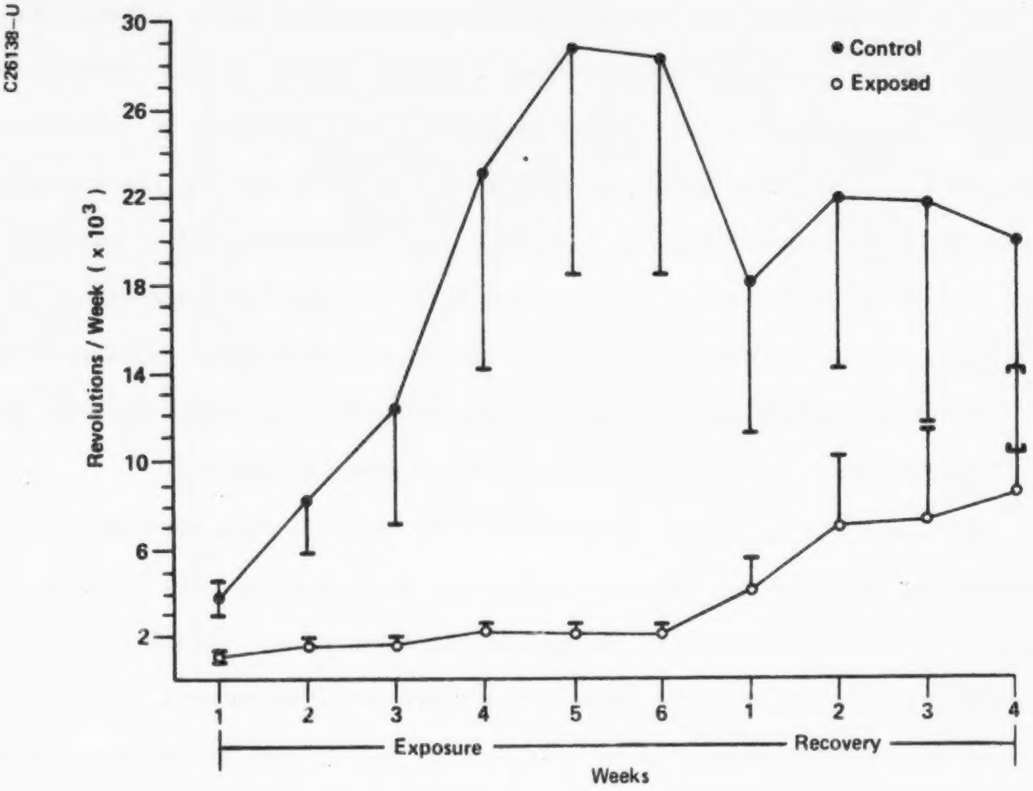

Figure 4.5. Effect of diesel exhaust on spontaneous locomotor activity in rats (Laurie et al., 1978)

diesel exhaust than by exposure to catalyst- and noncatalyst-treated gasoline engine exhaust (1:11 dilution). The causative agent(s) which affects rat behavior cannot be specified. It was postulated, however, that hydrocarbon components of diesel exhaust most likely accounted for the behavioral alterations observed.

4.2.4 Chronic Toxicity

The preliminary results of only two studies have been published thus far concerning the pulmonary damage resulting from chronic inhalation of diesel exhaust. These have employed high concentrations of emission products. Stuart and coworkers (1978) have exposed male rats (48 per group) to the fumes of an inefficiently operating diesel engine (50 ppm carbon monoxide, 10 mg/m^3 soot) alone and in combination with bituminous coal mine dust (6 mg/m^3) for six hours daily, five days per week for periods up to 20 months. Serial sacrifice and histopathologic examination of the lungs in rats inhaling diesel exhaust revealed particulate accumulations, vesicular emphysema, and beginning interstitial fibrosis. Inhalation of diesel exhaust together with coal dust produced similar alterations in the lungs as well as bronchiolar epithelial proliferation and inflammatory reaction. Carboxyhemoglobin levels were elevated in both treatment groups.

Parallel studies have also been conducted by Stuart and coworkers with Syrian golden hamsters exposed to diesel exhaust, and their results were summarized at a recent workshop (NIOSH, 1978). Exposures to diesel exhaust containing 4-6 ppm NO_2 and respirable particulates at 6-10 mg/m^3 were conducted five hours daily, five days per week, for up to 20 months. Histopathologic changes which resulted in the lungs included marked particulate aggregation in alveoli or macrophages, pulmonary consolidation, vesicular emphysema, interstitial fibrosis, and cuboidal metaplasia.

Very few studies have been conducted which involve chronic inhalation exposure to gasoline exhaust, usually because the carbon monoxide exposures involved are too great. Thus, direct comparison of histopathologic damage with that produced by diesel exhaust cannot be validly performed. In early reports of studies where mice were chronically exposed (>2 years) to diluted gasoline exhaust, most toxicologic observations were negative (Campbell, 1936). No adverse effects were noted on death rate, body weight, or rate of growth. When death occurred, it apparently involved heart failure accompanied by lung congestion. Pneumonia and pathologic alterations in the liver (congestion, atrophy with fibrosis or necrosis) were more common in exhaust-treated mice than in controls. More recent studies involving life-long exposure of rats to diluted (1:100) gasoline exhaust (containing 58 ppm CO and 23 ppm of total nitrogen oxides) have yielded several significant results (Stupfel *et al.*, 1973). The most important finding was the presence of bilateral renal sclerosis in more than half of the animals autopsied. In addition, a greater number of emphysematous lesions and spontaneous tumors of various organs were observed in exhaust-treated rats than in controls. However, no tumors of the respiratory tract were found. It is noteworthy that the levels of nitrogen oxides were very high in this study.

Most recently, a chronic study has been completed which involved the exposure of beagle dogs to raw or photochemically reacted gasoline engine exhaust 16 hours daily for 68 months (Hyde *et al.*, 1978). In all exposure groups, reversible lung damage was encountered. Dogs inhaling raw exhaust displayed hyperplasia of nonciliated bronchiolar cells, which apparently persisted long after cessation of exposure. With irradiated exhaust, incipient emphysema was produced, which was thought to result from exposure to nitrogen oxides (1.77 ± 0.68 mg/m^3 NO_2; 0.23 ± 0.36 mg/m^3 NO) and ozone (0.39 ± 0.18 mg/m^3).

4.2.5 Bioassays for Carcinogenicity

Pioneering work by Kotin and coworkers (1955) established that the particulate fraction of exhaust from an inefficiently operating diesel engine contains carcinogenic POM which are capable of producing tumors in experimental animals. Although the production of polycyclic hydrocarbons in an efficiently running diesel engine was extremely low, the exhaust from an inefficient diesel engine contained significant amounts of pyrene, benzo[a]pyrene, benzo[e]pyrene, benzo[ghi]perylene, anthanthrene, coronene, and an unidentified "compound X." Acetone solutions of benzene extracts from the particulate exhaust fraction from an inefficiently operating diesel engine were repeatedly applied (3 times weekly for more than 60 weeks) to the skin of mice (C57 black and A strain). A high incidence of skin cancers resulted in A strain mice when they were painted with particulate extracts obtained during full-load engine operation. These results corresponded with chemical analyses showing that polycyclic hydrocarbon emissions are greatest under conditions of full load and inefficient engine operation.

Kotin and coworkers noted that the diesel engine can be a greater source of polycyclic hydrocarbons than the gasoline engine (without oxidation catalyst) depending on engine operating conditions. However, previous studies by these same investigators demonstrated that benzene extracts of gasoline exhaust particulates are also capable of producing large numbers of skin cancers using C57 black mice (Kotin *et al.*, 1954).

Subsequent studies conducted on the potential carcinogenicity of diesel exhaust fractions have reportedly produced negative results. Clemo and Miller (1955) briefly mentioned that two fractions from diesel bus smoke yielded no carcinomas in mice, but experimental details were not reported. Mittler and Nicholson (1957) collected diesel and gasoline engine exhaust condensates (without using filters) and applied benzene extracts of this material twice weekly for eleven months to mice. They obtained a 76% incidence of skin tumors (presumably papillomas) in mice receiving a 4.0% gasoline exhaust extract, and no tumors in mice receiving a 2.26% diesel exhaust extract. These results are difficult to interpret, however, for several reasons: a) conditions of engine operation were not reported, b) the collection efficiency for the exhaust particulate fraction by the method employed is not known, and c) no chemical analyses were conducted on the engine exhaust condensates.

Thus far, attempts to produce tumors of the respiratory tract by the inhalation of either diesel exhaust (Stuart et al., 1978) or gasoline exhaust (Campbell, 1936; Stupfel et al., 1973) have not been successful. Nevertheless, the carcinogenicity of gasoline exhaust fractions by dermal application or subcutaneous injection in mice has been repeatedly confirmed (Wynder and Hoffmann, 1962; Brune, 1977; Pott et al., 1977).

Most investigators agree that the demonstrated carcinogenicity of particulate diesel and gasoline exhaust fractions, as well as particulate air pollutants from fossil fuel combustion, is largely (but not entirely) due to the presence of POM. Consequently, an intensive research effort has been mounted over the past several decades to thoroughly characterize the biological activity of chemicals in this class. A recent review of the published literature on POM indicated that benzo[a]pyrene is the most well-studied of all these compounds (Santodonato et al., 1978). Attention has focused on benzo[a]pyrene primarily because: a) it is an ubiquitous contaminant in atmospheric emissions from the combustion of fossil fuels, b) it is easily detected, and c) it is a potent animal carcinogen. It is evident that POM's in general and benzo[a]-pyrene in particular, when present in air, are nearly always found as adsorbed material on particulate matter. Thus, the inhalation of particulate matter from combustion processes can deliver a number of carcinogenic POM's, including benzo[a]pyrene, into direct contact with the respiratory tissues. Furthermore, numerous studies have indicated that the ability of suspensions of benzo[a]pyrene to induce experimental lung tumors can be considerably enhanced by concomitant exposure to particulate matter. It is suggested that particulates increase the carcinogenic response to benzo[a]pyrene by providing increased retention in the lung. In addition, simultaneous exposure to ciliastatic gases (e.g., SO_2, NO_2) can further enhance the respiratory tumor reponse to benzo[a]pyrene administration, possibly by inhibiting normal lung clearance mechanisms. Consequently, both gasoline and diesel exhaust are rightfully suspect as carcinogenic mixtures, and the amount of particulate material and POM's contained in them might be regarded as critical determinants of carcinogenic potential.

Nevertheless, in the absence of positive bioassay data involving chronic inhalation exposures, a direct comparison cannot be made between the carcinogenic potential of diesel versus gasoline exhaust. Based on the limited number of animal studies which have thus far been conducted, there are no data which suggest an increased carcinogenic threat from the substitution of diesel for gasoline exhaust.

4.3 Human Studies

4.3.1 Controlled Exposures

Little is known concerning the specific effects of diesel exhaust on various physiologic parameters in humans. Battigelli (1965) exposed volunteers to several dilutions of diesel exhaust containing 0.2-7.0 ppm NO_2, 0.2-2.8 ppm SO_2, 20-80 ppm CO, 900-15,000 ppm CO_2, 19.5-20% O_2, total aldehydes less than 1-2 ppm, and total hydrocarbons less than 5-6 ppm. Inhalation of the diluted exhaust for periods up to one hour had no effect on pulmonary resistance and produced no complaints of respiratory distress from the experimental subjects. Although the diesel exhaust produced no complaints when inhaled, the same subjects found that eye exposure to the diesel exhaust dilutions produced conjunctival irritation which often became intolerable.

4.3.2 Epidemiologic Studies

Attempts to establish an association between exposure to specific pollutants and adverse non-occupational health effects, especially cancer, are seldom successful. However, in occupational situations the effects of long-term exposure to high levels of a toxicant are more easily quantified; thus allowing for extrapolation back to the effect of small doses present in the ambient atmosphere. Unfortunately, only a few occupational studies involving diesel exhaust exposure have been published, and in none of these could the exposure be considered particularly intense in terms of concentration and/or duration.

4.3.2.1 Occupational Studies

Raffle (1957) authored a general review paper which made use of a number of examples to argue for the mutual benefits which can accrue to industry, medicine and the worker through the study of occupational and health records of employees. Absences from work because of sickness were shown to vary in frequency, duration and type depending upon the age, sex and occupational category of the worker. While acknowledging the diagnostic inaccuracy of individual absence records, Raffle emphasized the validity of comparisons between large groups.

Using records of the London Transport staff, he recalled the classical studies of coronary heart disease in conductors and drivers, in which uniform sizes at the times of initial employment provided critical information for the interpretation of self-selection. Raffle used health records of the conductors and drivers to show differences in their rates of absence attributed to "bronchitis" (undefined). Lastly, he reported on the incidence of cancer of the lung in relation to occupational exposure to the exhausts of diesel engine buses. When the London Transport staff, aged 45-64, were grouped according to expected low to high exposure there was no discernible gradient in the rates of death, retirement or transfer to alternative work during the period 1950 to 1954 due to lung cancer. There was an observed tendency for lung cancer death rates to follow the workers' residential patterns with respect to urban density and air pollution carried by prevailing winds. This is in agreement with the concept that "the amount of carcinogen in town air depends on the density of the population (possibly the number of coal fires) and that it is also driven by the prevailing wind." Thus, diesel exhaust could not be specifically implicated as a serious contributing factor. This clearly does not rule out possible health hazards of diesel engine exhausts, especially if diesel-powered urban vehicles were to predominate at some time in the future.

It could be argued, in connection with the London Transport staff work, that the period 1950 to 1954 was too early for any possible effects of diesel emissions to have become evident; diesel buses having been introduced gradually over a period from about 1935 to 1952. Therefore, the collection of lung cancer data has continued for this same group of workers, and by now a 25 year series is available (Raffle and Waller, to be published). The findings broadly support those for the first five years, and overall the lung cancer incidence rates are slightly lower than expected in the general population of London. This feature is common to other studies among occupational groups (Kaplan, 1959), and to some extent is may reflect the selection of relatively fit people for employment.

At about the same time as the paper by Raffle, air sampling data for two London Transport garages were reported by Commins and coworkers (1957). The Merton garage housed about 200 diesel buses and Dalston housed 120. Each garage was monitored from 6:00 P.M. to 7:00 A.M. on two nights: Merton in April and again in June, 1956; Dalston in October, 1956, and in June, 1957. Each 13 hour session was divided into 4 periods typified by fueling, down-time, departures and returns. Smoke samples for analysis of polycyclic hydrocarbons were taken at one site in each garage, and one outside (on the roof) as a control. A long-term smoke record (one week at Dalton and two weeks at Merton) was also taken at each garage.

On each occasion in every period, the average concentration of smoke was higher inside than outside each garage, though only slightly so during the down periods. Typically, the concentrations of hydrocarbons (pyrene, fluoranthene, 1:2- and 3:4- and 1:12-benzpyrene) were greater inside than out, but by a lower value than would be indicated by the inside to outside smoke concentration ratio. No marked excess of inside over outside sulfur dioxide concentration was observed. Variations in nitrogen dioxide concentrations followed the same pattern as smoke concentrations. Thus all pollutants were at their lowest levels in the down periods.

In their introductory remarks and at the conclusion of the report, the authors underscore the limited usefulness of their data in attempting to generalize: "The results are not to be applied without qualification to air pollution under the different conditions obtained in streets in the open air."

Kaplan (1959) analyzed the records of 154 lung cancer deaths among employees of the relief department of the Baltimore and Ohio Railroad from January 1, 1953 to December 31, 1958. Three groups, in order from greatest to least putative exposure, were compared: (1) those with direct occupational exposure to diesel or steam engine exhaust; (2) service workers or laborers in shops or roundhouses; (3) clerks and others rarely occupationally exposed. 96% of the employees were males and the few females belonged mainly to group (3) with low exposure. Group 3 was found to have a slightly greater age-adjusted rate of lung cancer deaths than group 1, while the rate for group 2 was considerably lower than for the others. Thus, the pattern did not conform well with the concept of exposure to diesel fuel or coal engine exhausts. Overall, the age-adjusted lung cancer death rate fell somewhat below that estimated for the United States male population.

Kaplan pointed out that these results were in strong conflict with those of a similar study by Heuper in 1955, i.e., that, while only 25% were operating employees (group 1), they accounted for 75% of the lung cancer cases. One of the reasons advanced for the relatively high lung cancer rate in group 3 was its greater proportion of urban dwellers. This factor was as least partly offset, however, by its higher proportion of females.

Not mentioned by the author, but clearly of concern is the limited character of the data. Selection in comparing local railway employees with the United States as a whole may operate through variations in the coding of causes of death due to lung cancer. In addition, the extent to which selection operated to retire ill employees was not evaluable. Lastly, it would have been desirable to consider causes of death which compete with lung cancer as well as to analyze medical records of the occurrences of chronic bronchitis and other illnesses, both of long and short-term character.

In a later review article Battigelli (1963) reported that none of the several measured components of air samples taken over several months in various studies of confined areas polluted by diesel engine exhausts (e.g., roundhouses, railway tunnels, bus garages) exceeded threshold limits established by the American Conference of Governmental Industrial Hygienists. He recognized, however, that in every major episode of air pollution with adverse effects to humans, none of the measured contaminants had exceeded accepted maximum concentrations.

Diesel exhaust was characterized as being distinct from the two major types of health-threatening polluted atmospheres typified by London and Los Angeles. Air polluted by diesel engine exhausts contains relatively low levels of carbon monoxide and of carcinogenic polycyclic hydrocarbons, as compared with gasoline engine polluted air. Diesel engines produce higher levels of objectionable odorants, conjunctival irritants and smoke, but Battigelli expressed the opinion that diesel exhausts were no more harmful than alternative forms of pollution.

The author pointed out that diesel engines discharge more nitrogen oxides and aldehydes per hour than comparable gasoline engines. This disadvantage was tempered by noting that the concentration of both of these gases in diesel exhaust is lower because of a greater air-to-fuel ratio in diesel motors. However, the air-to-fuel dilution factor is valuable only with respect to localized sources of pollution in confined spaces. When considering the environmental effects of a predominance of diesel powered vehicles, hourly discharges of nitrogen oxides and aldehydes would seem to be the more relevant issue.

In 1964, Battigelli teamed with two colleagues to report a cross-sectional study involving physical examinations and medical histories of 210 workers occupationally exposed for an average of 10 years to diesel exhausts in three Pittsburgh railroad engine houses. These workers were compared to 154 yard workers comparably distributed with respect to age, cigarette smoking histories, and extrapulmonary medical problems.

The internal air was sampled systematically at locomotive roof levels and at head-high "floor" levels at locations near to and distant from the locomotives. While NO_2, SO_2, total aldehydes, acrolein and hydrocarbons from C_1 to C_6 varied somewhat according to type of location, the general pattern indicated an extensive dilution of exhaust products. For example, the average concentration of total hydrocarbons was nearly identical for all three types of sample locations (engine roof, near floor and distant floor levels). Winter concentrations consistently exceeded those in summer.

The medical data were based on a physical examination, chest X-ray, electrocardiogram, spirometry, a standardized medical history with special focus on chronic respiratory diseases, and other standardized pulmonary function tests. The investigators reported that no significant differences were discernible between the 210 exposed workers and the 154 controls. In order to allay suspicions of insensitivity to existing major differences, they presented parallel comparative analyses of the workers who smoked versus those who did not smoke cigarettes within the previous 10 years. These analyses revealed consistently higher relative frequencies of dyspnea, cough and measures of bronchitis together with lower pulmonary function levels in cigarette smokers than in non-smokers.

Nonetheless, this study has shortcomings which resulted from an inability to pursue the original study plan. A low number of examinees was obtained, and participation was strictly on a volumtary basis. While the exposed workers were processed with a participation of better than 90% of the specific employed population, the non-exposed group showed a much smaller participation frequency. Thus, any attempt to carry out a formal analysis of statistical significance on a group of this composition is useless. However, based on their collected observations, particularly considering a mean exposure of 10 years, the fact that no major adverse effects were found should not be considered as trivial.

Several cohort mortality studies have been conducted with workers from underground mines in which diesel equipment was routinely used (Waxweiler et al., 1973; NIOSH, 1978). Although deaths attributed to malignant and non-malignant respiratory disease were elevated in certain instances, it was not possible to determine the potential contribution of diesel exhaust to this result. It was concluded that, "no excess mortality was attributable to the presence of diesel engines in some mines; however, there has probably been too little elapsed time for this observation to be definitive" (Waxweiler et al., 1973). Further epidemiologic studies involving metal and non-metal miners exposed to diesel exhaust are in progress (NIOSH, 1978) and may provide further clarification of the risk for lung cancer and respiratory disease.

A preliminary analysis of ventilatory function among 60 coal miners exposed to diesel emissions has recently been performed (Reger, 1978). Decrements in lung function over the period of a work shift could be demonstrated among miners exposed to coal dust either with or without concomitant exposure to diesel emissions. The decrements shown were no greater in the presence of diesel emissions than for exposure to ordinary mine atmospheres in the absence of diesel emissions.

The combustion of fossil fuels, resulting in exposure to POM, has long been associated with an increased cancer risk. More recently, however, inhalation of vehicular emissions has received attention as a specific factor in the etiology of malignant disease. In this regard it was reported that moderately elevated relative risks for cancers of the nose, pancreas, and prostate were observed in mechanics and repairmen (Viadana et al., 1976). In addition, bus, taxi, and truck drivers showed increases in cancer of the pancreas; locomotive engineers were at increased risk for lymphomas and cancers of the buccal cavity and pharynx. Other investigators observed an elevated lung cancer rate in Los Angeles County for workers in the auto repair industry (Menck and Henderson, 1976).

Occupational exposure to gasoline engine emissions have also been of interest to epidemiologists for the development of non-malignant disease. One recent report concerns a retrospective cohort study of mortality among approximately 1600 motor vehicle examiners employed in New Jersey during the period 1938 through 1973 (Stern and Lemen, 1978). Because previous studies had shown that chronic low-level exposures to carbon monoxide may exacerbate coronary heart disease, measurements were made of carbon monoxide levels (average concentration 22 ppm) and heart disease mortality was specifically pursued. The study, however, failed to show a significant excess of heart disease deaths, but instead revealed a significant excess of cancer deaths (13 observed vs. 6.69 expected) for individuals with greater than 30 years since onset of employment. The excess of cancers was not associated with a particular organ site. Since cause-specific mortality rates for the general United States white male population were used for comparisons in this study, the consistently higher New Jersey cancer death rate was not taken into consideration. Thus, it is not possible to conclusively attribute the excess of cancer deaths in this study to occupational exposure to automotive exhaust.

A study of 386 children who died of malignant disease in the province of Quebec during the years 1965 through 1970 has suggested a correlation with specific types of occupation of the father (Fabia and Thuy, 1974). Prior to undertaking the study it had been postulated that the risk of malignant disease may be greater among children whose fathers are occupationally exposed to petroleum products. The study revealed that excess cases of cancer existed among the children of motor vehicle mechanics, machinists, miners, and painters. These striking results do not provide an explanation of the mechanism by which such a phenomenon might occur. Nevertheless, in other studies involving occupational exposure to known carcinogens (e.g., vinyl chloride) it has been reported that fetal deaths and congenital defects may be increased in the offspring of exposed fathers (Infante et al., 1976; U.S. Public Health Service, 1976).

4.3.2.2 Community Studies

A suitable community population has not yet been studied which might reveal the impact of diesel emissions in the ambient atmosphere on human health. However, the effect of diesel emissions on the general population may be too small to be accurately detected and separated from other variables. Similarly, the question of whether gasoline engine emissions represent a significant carcinogenic threat to man has not yet been resolved (Lawther and Waller, 1976). This is primarily due to the diversity of pollutants in the environment, and the inability to identify a suitable population having significant exposure to a specific pollutant type. Nevertheless, a report implicating emissions from automobiles (presumably gasoline-powered) as an important cause of cancer has been published in Switzerland (Blumer *et al*., 1972). In this study of a very small population, deaths due to cancer of various sites were nine times more frequent (11% *vs*. 1.2%) among residents of a Swiss mountain town living near a highway as compared to residents living in an area remote from traffic. The authors concluded that differences in age, occupation, exposure to non-automotive combustion products, sex, and smoking habits could not entirely account for the increased cancer mortality rate. However, the population studied was too small to allow for determination of age/sex/site specific cancer rates. Therefore, the reported observations may have little overall significance.

In a follow-up study to clarify these observations, it was found that soil content of polycyclic aromatic hydrocarbons in this region showed a correlation with proximity to a highway (Blumer *et al*., 1977). The compounds identified were present as a complex mixture of unsubstituted three- to eight-membered rings and heavily alkyl-substituted derivatives, which the author concluded had originated primarily from automobile exhaust. Thus it was believed that the observed mortality from cancer in this area might indeed be associated with exposure to automotive exhaust. Little can be said, however, concerning the levels of exposure to automobile-derived carcinogens among those residents living near the highway. Since the Swiss town was situated in a deep valley with frequent thermal inversions, it is likely that these persons had unusually high exposures to exhaust emissions. On the other hand, deaths due to non-malignant disease (heart and circulatory) did not seem to vary greatly between roadside and non-roadside residents.

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5.0 Identification of Knowledge Gaps

5.1 Biological Effects

There are no areas concerning the biological activity of diesel emissions in which complete information is available. These gaps in the health effects data base are a reflection of the limited number of studies which have been conducted, rather than an indication that the data are not obtainable. Nevertheless, this lack of information is sufficient to prevent the formulation of any definitive health risk assessment at this time. Recognizing that data in certain areas are more valuable than in others for the purpose of risk assessment, selected gaps in our body of knowledge are discussed (but not necessarily in order of priority) below.

A population study has not yet been conducted in which a diverse human group has been studied with respect to chronic low-level exposure to diesel emissions. Therefore, nothing can be said regarding the existence of susceptible groups, effects on mental and physical development, interference with reproductive success, exacerbation of pre-existing disease, or interaction with other common environmental pollutants.

Data derived from human exposure studies are of paramount importance in establishing a chemical threat to health. Unfortunately, the available epidemiologic evidence is insufficient to define the effect of diesel emissions on human populations. The historical epidemiologic literature regarding morbidity and mortality has not adequately addressed the factors of exposure intensity and duration, contial populations, and possible bias in reporting of results. The comparative analysis of diesel emissions health effects is further complicated by uncertainty over the consequences of human exposure to gasoline engine exhaust. Moreover, the impact of catalyst-treated gasoline engine exhaust on human health is virtually unknown.

Thus far only one investigator has examined the clinical symptoms and metabolic alterations produced by controlled human exposures to diesel exhaust. Numerous questions still remain to be answered including: lung deposition, retention and clearance of diesel particulate; alterations in enzyme activity; effects on hematologic parameters; and correlation of physiologic effects with concentration of specific components in the diesel exhaust mixture.

Animal bioassays conducted with diesel exhaust have provided important data regarding the production of histopathologic lesions and biochemical alterations in the lung, behavioral disturbances, susceptibility to infection, effects on the heart, and decrements in pulmonary function. However, little is known concerning the reversibility of this damage or the correlation between extent of tissue damage and degree of functional impairment. In addition, dose-response studies have not been conducted, nor is it known whether the toxic effects produced by subchronic exposures are indeed life-shortening.

Recognizing that the lung is the primary organ which contacts airborne diesel emissions, several parameters of its interaction with diesel exhaust components should be classified. Furthermore, it is difficult to relate morphologic alterations to biochemical events produced in response to toxic insult. Since the respiratory epithelium is a major site for the systemic absorption of airborne chemicals, we should also know the extent to which diesel particulate and its adsorbed materials (e.g., carcinogenic POM) reach the systemic circulation via the lung. This is especially relevant in light of studies implicating automobile exhaust as a contributing factor in cancers of various internal organs.

Thus far it has not been possible to attribute most of the toxic effects of diesel exhaust to the action of specific components in the mixture. Likewise, a similar problem exists with most studies involving exposure to gasoline engine exhaust, and thus comparisons between the two systems are difficult.

The behavior of environmental pollutants in isolated organs and individual cells often provides critical data concerning mechanisms of toxic action. Numerous in vitro studies with extracts of airborne particulate pollutants and their individual components (e.g., POM) have revealed the basis by which damage is produced. With diesel emissions, however, nothing is known regarding cytotoxicity, interaction with critical cellular macromolecules, cell transformation, or cytogenetic damage.

6.0 Recommended Research

6.1 Biological Effects

Animal studies conducted with diesel exhaust have revealed a diversity of toxic effects in several species. These results are sufficient to warrant further investigations in several areas. A list of suggested biological research projects is presented below which would fill many of the information gaps identified in Section 5.1. This, however, is not a list of research priorities.

1. Conduct of occupational and community-based epidemiologic studies to provide morbidity and mortality data regarding diesel exhaust exposure - Cancer as a biologic endpoint should be of primary interest, but chronic respiratory disease such as emphysema must also be carefully evaluated. Whenever possible, information should be obtained regarding current and past employment, respiratory symptoms, smoking histories, and other health information such as genetic factors. For morbidity studies, a battery of tests which includes pulmonary function measurements and sputum cytology should be conducted. In addition, comparison of the health status of persons exposed to gasoline engine emissions with those exposed to diesel emissions is highly desirable. Furthermore, a means to quantitate observed exposures to diesel exhaust will be necessary for the formulation of valid health risk exposure criteria. It must be recognized, however, that there are few opportunities for separating diesel and gasoline exposure.

2. Cytogenetic testing of workers having high occupational exposures to diesel emissions - Analysis should be made for chromosomal aberrations in cultured lymphocytes taken from exposed workers. These determinations are felt by many scientists to provide an indication of increased cancer risk and potential for transmission of birth defects and mutations. The use of somatic cells to predict a mutagenic effect that may occur in germinal cells is not entirely valid. Nevertheless, numerous examples of the positive correlation between a chemical's ability to produce chromosome aberrations in somatic cells and its carcinogenic/mutagenic activity indicate that cytogenetic data should be carefully evaluated. Moreover, the widely held view that cancer arises as a result of somatic mutation emphasizes the need for cytogenetic testing.

3. Conduct of inhalation exposure studies in animals using various dose levels - These studies should be designed to detect dose-response parameters and ascertain the reversibility of treatment-induced damage. Included in the experimental protocols should be means to detect neurotoxicity, reproductive effects, cardiovascular function, effects on host defenses and threshold dose levels for toxic response.

4. Evaluation of diesel exhaust mutagenicity in vivo and in vitro - For the detection of gene mutations the use of mammalian somatic cells in culture (with and without metabolic activation) should be employed. Chromosomal aberrations should be measured by in vivo cytogenetic tests in animals, dominant lethal effects in rodents, and heritable translocation tests in rodents. Primary DNA damage should be detected using tests for unscheduled DNA repair synthesis and sister chromatid exchange in mammalian cells (with and without metabolic activation), DNA repair in bacteria, and mitotic recombination and/or gene conversion in yeast.

5. Evaluation of in vitro carcinogenesis by diesel emissions - Previous studies have shown that organic extracts of certain samples of airborne particulate matter can transform mammalian cells in culture. To establish the presence of carcinogenic materials in diesel exhaust particulate, various systems could be employed such as early passage hamster embryo cells, baby hamster kidney cells, C3H10T 1/2 mouse fibroblasts, and several organ culture systems using respiratory tract epithelium. Results from such tests would provide important data to support the observed mutagenic effect of diesel particulate extracts in the Ames assay.

6. Evaluation of in vivo carcinogenesis by diesel emissions - Studies using various fractions of diesel exhaust and employing various routes of administration should be pursued. In addition, an evaluation of the potential involvement of cocarcinogens should be conducted.

7. Evaluation of the pulmonary deposition, clearance, and transport of diesel particulate - Animal models should be employed to ascertain the fate of inhaled diesel particulate matter.

8. Evaluation of the toxicity of the vapor phase components of diesel exhaust emissions - Standard inhalation experiments in animals using reconstituted mixtures of gaseous components at ratios found in raw exhaust should be conducted.

9. Evaluation of the effect of variation in fuels and engine operating parameters on the resultant toxicity of diesel exhaust emissions - These parameters can be incorporated into nearly all of the suggested research studies listed above.

10. Conduct of parallel toxicity studies with diesel and gasoline engine exhaust - Using identical test conditions, a comparison should be made between the relative hazards to health of diesel versus gasoline engine exhaust. These studies would provide important data concerning the ultimate environmental impact and public health implications of a major changeover to the use of diesel vehicles.